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Department of Health and Environmental Sciences
STATE OF MONTANA HELENA, MONTANA 59601

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Director

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To All Interested Parties:

Attached is the working paper no. 3 on urban pollutants, including photochemical oxidants, hydrocarbons, nitrogen oxides, and carbon monoxide.

The first three of these pollutants are grouped together, because they all enter into photochemical reactions.

The fourth pollutant, carbon monoxide, is considered separately because it does not participate in the photochemical process although it is an urban pollutant.

As in the earlier working papers, we ask that interested parties point out any shortcomings in the data or analysis. Any new information produced by public comment, plus economic and environmental considerations, will be used in determining what standards should be proposed to the Board of Health and Environmental Sciences.

All this information plus that generated for all the other pollutant groups will be included into a draft environmental impact statement to be issued in October. An adequate period for public comment will follow prior to issuance of the final statement and the commencement of hearings before the board.

As it now stands, this working paper is considered a draft, and may have some rough spots. The research results, however, are considered valid and representative of the body of scientific knowledge related to the pollutants discussed.

We invite comments and suggestions that would improve this paper.

Although the issuance of working papers is behind the schedule envisioned in working paper no 1, we still expect to meet the original deadline set for issuance of the draft impact statement.

Sincerely,

Michael D. Roach,
Chief, Air Quality Bureau

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Montana Ambient Air Quality Study

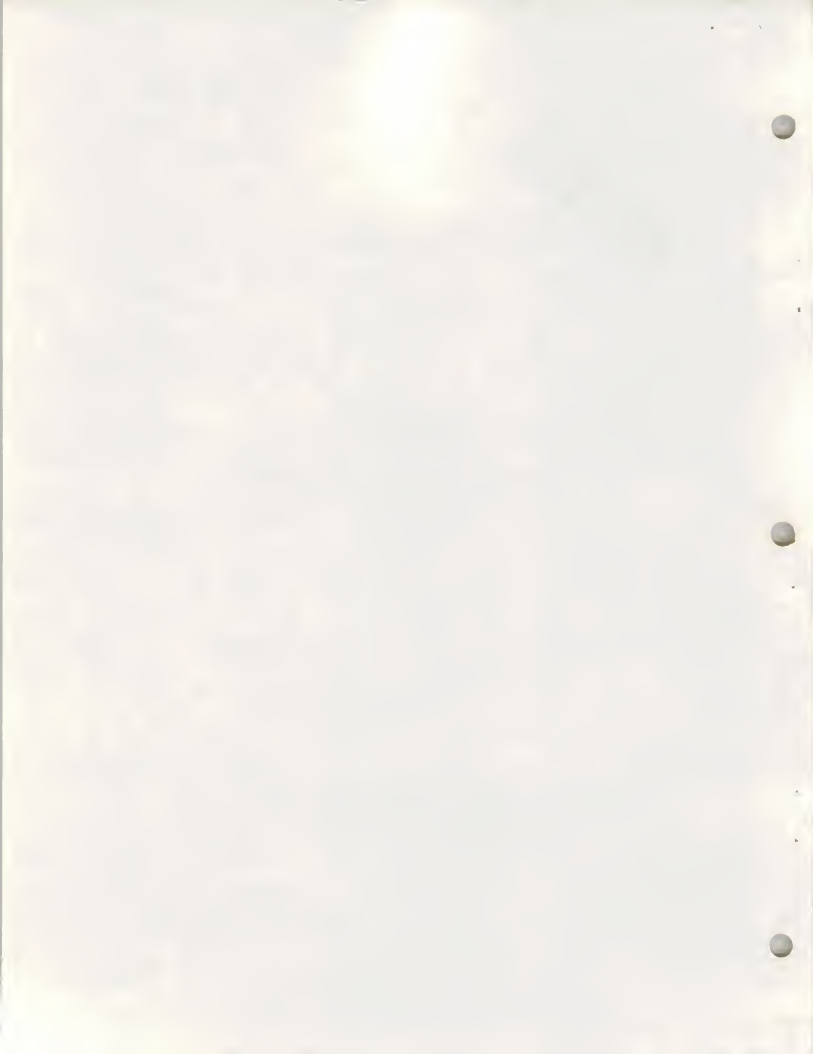
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PHOTOCHEMICAL OXIDANTS, HYDROCARBONS, NITROGEN OXIDES, AND CARBON MONOXIDE

Working Paper Number Three for Developing
Enforceable Ambient Air Quality Standards
for Montana

State of Montana
Department of Health and Environmental Sciences
Environmental Sciences Division
AIR QUALITY BUREAU

August, 1978



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INTRODUCTION

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Photochemical pollutants represent a class of compounds that are products of atmospheric reactions involving hydrocarbon pollutants*, nitrogen oxides, oxygen, and sunlight. These compounds originate for the most part from human activities and are highest in the vicinity of large urban areas. The toxic products of photochemical reactions consist mainly of ozone, nitrogen dioxide, and peroxyacetylnitrate (PAN), with lesser amounts of other peroxyacetylnitrates and peroxy-compounds, referred to collectively as oxidants.

The mechanism by which photochemical smog is produced is extremely complicated and not completely understood. Researchers have found that a minimum level of sunlight is necessary to initiate the photochemical sequence. Further, the photochemical reactions are dependent on critical levels of hydrocarbons and nitrogen oxides. If either of these compounds is not present in sufficient amount, the photochemical reaction sequence will not be completed and no significant photochemical products will be produced.

Figure 1 depicts the chemical changes that are observed to occur when a mixture of hydrocarbons and nitrogen oxide pollutants is exposed to sunlight under conditions capable of causing photochemical oxidant production. The curves of Figure 1 indicate the overall photochemical process as consisting of two distinct reaction stages occurring consecutively. During the first stage, nitric oxide is converted into nitrogen dioxide without any appreciable buildup of ozone or other non-nitrogen dioxide oxidants. The second stage starts when almost all of the nitric oxide has been converted into nitrogen dioxide and is characterized by rapid accumulation of ozone and other oxidant and nonoxidant products.

* The term hydrocarbon is meant to include all hydrocarbon organics capable of participating in the atmospheric oxidant formation process. Hydrocarbons and organics are used interchangeably in this document.

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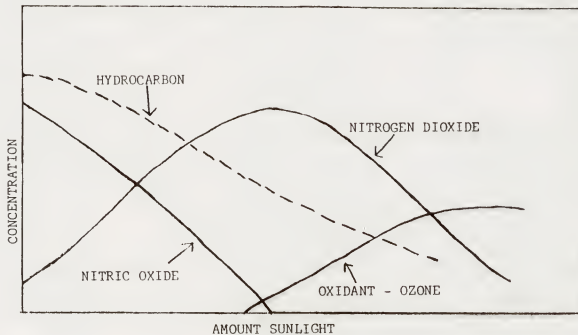


Figure 1. Chemical changes occurring during exposure of hydrocarbon-nitrogen oxide-air systems to sunlight

The relative amounts of photochemical pollutants differs from one area to another. The amounts present in a particular locale depend on the initial concentration of hydrocarbons, nitrogen oxides and sunlight. The amount of total oxidant could be the same in two cities although the proportion of the various precursors would be quite different. The potential effects of any photochemical reaction thus depends on which compounds are created in the reaction and is in turn dependent on the amount of each precursor compound present in the atmosphere.

Historically, the term photochemical oxidants has referred to substances producing specific types of chemical changes defined with respect to the potassium iodide measurement method. In this paper three products of photochemical reactions are evaluated: ozone (O_3), peroxyacetylnitrate (PAN), and nitrogen oxides (NO_x). Nitrogen oxides are included even though elevated levels of these pollutants come directly from industrial sources. They are both precursors and products of photochemical reactions. Hydrocarbons, because of their importance in the photochemical generation of ozone, also are analyzed here. Currently there are federal ambient standards for ozone and nitrogen oxides (as nitrogen dioxide). A federal ambient standard also has been promulgated for hydrocarbons but is interpreted as a guideline for planning purposes rather than an enforceable standard.



Chapter I

OXIDANTS

Introduction and Summary

Ozone is the principal pollutant discussed in this chapter. Peroxyacetylnitrate (PAN) is included when it impacts the environment at or near levels known to occur in ambient air.

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Photochemical reactions involving man-made organic compounds and NO_x emissions are the major source of ozone and PAN in urban areas. Natural sources of ozone include intrusions from the natural ozone layers of the stratosphere, and photochemical reactions between natural organic compounds and NO_x .

The effects of ozone are many and varied. There is strong scientific evidence indicating detrimental effects from ozone in humans, agricultural plants, ecosystems, and materials at or near the 0.1 ppm level over various exposure periods. To prevent these effects, the Environmental Protection Agency (EPA) has set a national ambient standard for ozone of 0.08 ppm for 1 hour, not to be exceeded more than once per year. Currently this value is being revised by the EPA to 0.1 for 1 hour for the primary standard and 0.08 ppm for the secondary standard.

Because of the complexity of the atmospheric reactions leading to ozone generation, control of ozone has proved difficult. Currently, cities in Montana and elsewhere regularly exceed the standard during the summer when conditions are favorable for photochemical generation of ozone.

The recommended method for monitoring oxidants measures only ozone because of its predominance among the group of photochemically generated compounds (excluding NO_2). Levels of ozone indicate the total amount of oxidant, some of which have not been identified as to chemical composition and environmental impacts. Since ozone is only a part of the total oxidant mix, measurements of ozone alone may not accurately reflect the total impact of photochemical oxidants.



OZONE POLLUTION IN MONTANA

Sources

Ozone/oxidants are generated from a number of light initiated chemical reactions. The formation of oxidants is the net result of two processes acting together: 1) a physical process encompassing dispersion and transport of the oxidant precursor emissions (hydrocarbons and NO_x), 2) the photochemical reaction process. Both processes are strongly influenced by meteorological conditions such as wind, solar radiation, temperature, and humidity.

Photochemical pollutant formation occurs predominantly during the summer. Laboratory studies have found that at a temperature below 55° to 60° F atmospheric reactions between precursor emissions are not fast enough to generate problem levels of oxidants.

Transport of photochemical precursors is important in determining the amount of pollutant products ultimately resulting from interaction of the precursor emissions. The types of precursor transport fall into three categories: 1) short-range transport downwind from urban centers, 2) transport in the form of urban plumes extending as far as 100 miles downwind from the source, and 3) long-range transport of several hundred miles associated with high pressure weather systems. Because of these transport phenomena, photochemical pollution is viewed as having widespread health and welfare implications.

Finally, recently compiled statistics on windspeed and pollutant mixing heights show that limited dispersion episodes favor photochemical oxidant buildup. The common areas for limited dispersion in Montana are in the Rocky Mountain portions of the state.

Although Montana does not possess the major population centers found in other states, it has populated areas with severe air stagnation episodes and enough sunlight to cause photochemical pollutant buildup in the summer.

Elevated ozone also results from the photochemistry of natural organic

and NO_x emissions and from stratospheric intrusions of ozone to ground level. Based upon evidence from global circulation patterns and data on ozone concentrations and variations in rural areas, ozone levels from natural sources are estimated to range from 0.02 ppm to 0.05 ppm in the vicinity of 45° N latitude, which forms the southern border of Montana. The highest hourly ozone concentrations from nonindustrial sources can be near or even above 0.08 ppm (present federal standard) and most likely occur during March, April, and May. During the summer, when urban and industrial emissions produce the highest ozone levels, natural ozone levels are estimated to be 0.03 ppm. It is important in establishing a monitoring network to know the amount and duration of natural elevated ozone levels. Only then can man-caused ozone pollution be adequately quantified and a realistic control strategy devised.

Ambient Data

Ozone monitoring currently is being carried out in three Montana cities and at several rural locations. Data from these sites are used to determine whether federal standards are being violated and to indicate the total photochemical complex of which ozone is the predominant component.

Data for Billings and Missoula are given in Table 1. The Billings data indicates ozone violations occur predominantly during warm weather periods with most frequent violations of the present 0.08 ppm federal standard and proposed new 0.10 ppm federal standard during the summer. The highest ozone level recorded for Montana (1975-1978) was found at the Billings station. The value of 0.160 recorded there in July 1975 is approximately twice the present federal standard.

The rural locations where ozone monitoring is currently being conducted are at Colstrip and Ft. Howes (EPA site) in the southeastern portion of the state. Neither is included here because of equipment difficulties at Colstrip and unavailability of data from the EPA for the Ft. Howes location. In 1975,

TABLE 1 OZONE AMBIENT DATA (ppm)

BILLINGS

Month & Year	Total Readings	1 Hr. Periods Over:						0.10**	0.16
		0.01	0.02	0.04	0.06	0.08*			
April 1978	499	398	325	20	0	0	0	0	0
March 1978	725	422	350	65	9	4	2	0	0
February 1978	659	431	361	45	4	1	0	0	0
January 1978	672	335	273	2	0	0	0	0	0
December 1977	354	264	0	0	0	0	0	0	0
November 1977	704	289	211	8	0	0	0	0	0
October 1977	327	126	89	1	0	0	0	0	0
September 1977	586	249	199	20	0	0	0	0	0
August 1977	579	377	316	61	1	0	0	0	0
July 1977	681	518	487	93	0	0	0	0	0
June 1977	644	434	396	130	4	0	0	0	0
April 1977	552	383	343	81	0	0	0	0	0
March 1977	718	435	369	12	0	0	0	0	0
February 1977	643	249	198	1	0	0	0	0	0
December 1976	673	173	93	0	0	0	0	0	0
November 1976	651	162	114	0	0	0	0	0	0
October 1976	721	192	103	0	0	0	0	0	0
September 1976	645	230	157	10	0	0	0	0	0
August 1976	697	377	322	68	10	2	0	0	0
July 1976	718	554	522	237	32	9	1	0	0
June 1976	597	406	359	51	2	1	0	0	0
May 1976	660	464	388	23	0	0	0	0	0
April 1976	615	472	432	128	27	1	0	0	0
March 1976	714	503	470	182	0	0	0	0	0
February 1976	630	373	329	14	0	0	0	0	0
January 1976	716	322	224	0	0	0	0	0	0
December 1975	720	218	124	0	0	0	0	0	0
November 1975	540	178	121	0	0	0	0	0	0
October 1975	722	251	126	4	0	0	0	0	0
September 1975	286	106	82	5	0	0	0	0	0
July 1975	533	411	393	304	157	50	16***	0	0
June 1975	694	498	426	336	138	6	0	0	0
May 1975	684	490	458	348	152	22	3	0	0

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Table 1 continued

BILLINGS

		Total	1 Hr. Periods Over:						
Month & Year		Readings	0.01	0.02	0.04	0.06	0.08*	0.10**	0.16
April	1975	698	611	592	514	280	28	0	0
March	1975	683	546	526	343	111	0	0	0
February	1975	667	463	424	190	24	0	0	0
January	1975	698	351	298	75	7	0	0	0

MISSOULA

March 1978	646	391	346	89	1	0	0	0
February 1978	659	254	191	40	0	0	0	0
January 1978	621	98	61	0	0	0	0	0
December 1978	37	17	6	0	0	0	0	0

WOLF POINT 1975 2160 Total readings:

Average yearly ozone average : 0.028 ppm No 1 hr. periods over 0.08 ppm

* Violations of Federal primary and proposed secondary 1 hour 0.08 ppm ozone standard.

** Proposed federal primary 1 hour 0.10 ppm ozone standard.

*** Highest value recorded for Montana - 0.160 ppm.

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monitoring for rural ozone levels was conducted by Research Triangle Institute (Coffey et al, 1977) at Wolf Point Montana. The annual value of 0.028 ppm with no excursions over 0.08 ppm indicates low background levels for this rural Montana location.

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HUMAN HEALTH EFFECTS

Three lines of experimental evidence are presented in this section to illustrate the impacts of ozone and other photochemical substances on human health: 1) animal toxicological studies 2) clinical studies 3) epidemiological studies.

Animal toxicological studies are valuable for determining effects of certain pollutants on laboratory animals under controlled conditions in experiments that may not be ethically justifiable on human subjects. Although the findings of these studies are not directly applicable to humans, there is a general understanding that responses found in experimental animals most likely will cause similar effects in humans. For more direct application to humans, clinical health experiments using low level short term exposures allow reliable evaluation of human dose-response relationships for both single and combined oxidants.

The epidemiological studies evaluate health responses under ambient conditions common to the human environment. Results from epidemiological studies provide the best information regarding the effects of oxidants on human health, although pollutant concentrations and exposure durations are extremely difficult to quantify in these studies. Taken together these three experimental techniques allow a thorough evaluation of ozone effects on humans.

ANIMAL STUDIES

There is a broad range of evidence from animal studies that suggests increased susceptibility to bacterial infection following ozone exposure. Some of these studies indicate 0.08 ppm (Coffin et al, 1968) and 0.1 ppm (Miller et al, 1977) ozone is capable of causing increased mortality in strains of mice inoculated with

bacterial aerosols. Coffin and Blommer (1970) found that preexposure to low temperatures increased mortality in animals exposed to bacteria and subjected to 0.07-0.09 ppm ozone for 3 hours. Other experiments exposing mice to various combinations of pollutants such as 0.05 ppm ozone and 2 ppm NO₂ for 3 hours, (Ehrlich et al, 1976) or 0.1 ppm ozone, and 0.9 mg. sulfuric acid (H₂SO₄)/M³ for 2 hours (Gardner et al, 1977) showed an additive effect. Lowering the concentration necessary to cause increased mortality in mice after bacterial infection. These studies demonstrate that basic defense mechanisms common to all animals and humans are disturbed at ozone levels common in certain urban areas.

Morphological abnormalities within the lungs and pulmonary system of experimental animals occur after chronic long term exposure to ozone. Pan et al, (1972) found rabbits exposed to ozone at 0.4 ppm 6hr/day, 5 day/wk, for 10 months showed emphysematous and vascular-type lesions in the lungs along with pulmonary artery thickening. At 0.54 ppm ozone continuous exposure for 3 months Stephans et al, (1976) found emphysematous and fibrotic changes and thickening of alveolar septa and an increase in the number of alveolar macrophage in laboratory rats. These studies suggest similar effects are possible in humans after chronic exposure to ozone.

There are two types of physiological response to ozone exposure; adaptation/stress and pathological. Biochemical studies provide evidence that no absolute demarcation can be made between these two responses. It is generally accepted that near levels of .1 ppm ozone several types of adaptive response are seen while 0.5 ppm ozone exposure has toxic potential in cells (NAPCA, 1970). Adaptation and stress have a valuable biological function in all animals. However, stress without adaptation, especially in the long term, can be associated with premature mortality, morphological changes, and predisposition to infection. Stress could be considered an unwarranted and adverse impact from ozone exposure.

There is evidence suggesting that ozone may have mutagenic properties in living cells. Zelac et al, (1971) exposed Chinese hamsters to 0.24 ppm ozone for 5 hours

and found a significant increase in chromosome breaks in circulating blood lymphocytes. Chromosome breakage also has been found in human cells exposed in vitro to ozone (9.0 ppm) for 5-10 minutes. (Fetner, 1982)

CLINICAL STUDIES

There is conclusive evidence indicating a relationship between ozone exposure and reductions in mechanical function of human lungs. Recent studies have shown statistically significant lung function changes in healthy smokers and nonsmokers at concentrations of 0.25 to 0.78 ppm alone and in combination with other oxidants for 2 hour exposures under intermittent exercise conditions (Bates and Hazucha, 1973; Hazucha et al, 1973). Research by Von Nieding et al, (1976) suggests detrimental lung changes may be significant to healthy persons for a 2 hour ozone exposure in the range of 0.15-0.25 ppm. Some individuals more sensitive to ozone appear more susceptible to ozone induced lung function changes. During exposure of a group of persons to ozone, Goldsmith and Nadel (1969) found one individual with greater than normal airway resistance who showed decreased performance at 0.1 ppm ozone for 1 hour.

There is conflicting evidence on the response in lung function found with repeated exposure to ozone. Hackney et al, (1977) reports that lung function decline seen in some individuals was relieved somewhat following repetitions of 0.5 ppm ozone exposure 2 hours each day for 4 days. Contrary evidence was found in previous work by Hackney et al, (1975) which showed that individuals with minimal lung dysfunction after one exposure to 0.5 ppm ozone for 2 hours still showed greater dysfunction after a similar exposure on the following day.

Human lung function also appears susceptible to reduction by combinations of pollutants. Studies by Hackney et al, (1975) have shown that a combination of 0.5 ppm ozone, 0.3 ppm nitrogen dioxide, and 30 ppm carbon monoxide for 4-5 hours caused marked changes in respiratory functions in sensitive individuals. In an experiment conducted by Hazucha and Bates (1975) results showed a greater than additive effect in lung dysfunction when ozone and sulfur dioxide were

administered together. This experiment emphasizes that individuals subjected to urban air containing many pollutants may be affected by ozone in combination with other pollutants at levels lower than the dose necessary to cause effects when ozone is administered alone.

The most obvious reaction of man exposed to oxidant air pollution is the development of eye irritation. In experimental studies, ozone at concentrations often found in the ambient air of U.S. urban areas, has not been found to cause eye irritation (Shuck and Boyle, 1959). The constituents of the oxidant complex that are presently believed to cause eye irritation, and in some cases tears are peroxyacetylnitrate (PAN), peroxy benzoylnitrate, acrolein, and formaldehyde (National Academy of Science (NAS), 1976).

EPIDEMIOLOGICAL STUDIES

Short term

The effect of oxidants on lung function in sensitive persons is not entirely conclusive but indicates risks at the 0.15 to 0.25 ppm ozone level. A study by Schoettlin and Landau (1961) showed that asthmatics in Los Angeles had a significantly greater number of attacks on days when daily maximum oxidant levels exceeded 0.25 ppm than on days when they were below this level. Motley *et al.* (1959) found individuals with chronic respiratory disease had reduced residual lung volume and lung function at maximum oxidant concentrations of 0.20 to 0.53 ppm calculated as ozone. Maximum concentrations of other pollutants measured in this study fell into the following ranges: 1) NO_x , 0.2-0.6 ppm 2) SO_2 , 0.05-0.25 ppm 3) CO , 5-27 ppm.

Children have been identified as particularly susceptible to lung function changes from oxidants. Kagawa and Toyana (1975), in a study of Japanese school children found that 25% of the students sampled had lung function changes and increased airway resistance correlated with high oxidant and SO_2 levels. Oxidant concentrations ranged from 0.01 to 0.30 ppm during the experiments. This study also found that ozone was the most highly correlated (of all the oxidants

measured) with pulmonary function changes. Kagawa and Toyana's work provides some epidemiological verification to the clinical studies on ozone-lung function relationships.

Short-term oxidant exposures have been positively associated with the promotion of illness in healthy populations. Increased cough and chest discomfort has been observed in a study of Los Angeles student nurses conducted by Hammer et al, (1974) at a daily maximum hourly oxidant concentrations of 0.25-0.29 ppm. Head-aches and eye irritation were found to occur at still lower levels, increasing with exposure to oxidants at 0.15-0.19 ppm maximum hourly average.

Recent Japanese studies (Shimizu, 1975) found increased rates of cough, chest discomfort, eye irritation and other symptoms at oxidant concentrations between 0.10 and 0.15 ppm. Furthermore, data from these studies found eye irritation was highest in the morning when ozone concentrations were low while elevated chest discomfort was associated with high afternoon ozone level measurements. These results seem to indicate that lung problems are predominately ozone related while eye irritation results from other constituents of the photochemical mix.

Short term oxidant levels have been consistently associated with decreases in exercise performance. Wayne et al, (1967) found reductions in the performance of high school cross-country runners to be correlated with oxidant concentrations over a range of 0.03 to 0.30 ppm. Folinsbee (1975, 1977) suggests that the decreased performance is the result of ozone acting as a irritant to the respiratory system. Irritation was found to result in a reduction in the respiratory capacity and total volume of human lungs.

Recently, DeLucia and Adams (1977) have shown that exercising persons experience chest discomfort and reduction in pulmonary function at a level of 0.15 ppm ozone for a one hour exposure period. These results indicate that individuals engaged in strenuous exercise are more sensitive to the adverse health effects of ozone than nonexercising individuals and should be considered a susceptible portion of the population when an ambient oxidant standard is being set.

Long term

Studies into the response of human subjects to long term oxidant exposure are not as extensive as short term investigations. This is partly because of the difficulty of doing accurate studies of this kind and the relatively brief time since elevated ambient ozone levels have become a problem.

Several studies suggest a relationship between oxidant exposure and chronic respiratory morbidity. Goldsmith and Deane (1965) and Linn (1976) have observed a parallel between historically high oxidant levels in Los Angeles and persistent cough and phlegm in telephone workers and a group of women there as compared to lesser affects among counterpart groups in San Francisco, where ozone levels are lower. Although both experiments indicate a response to long term oxidant levels, the study designs did not allow specific oxidant levels to be associated with contribution toward respiratory disease. Peters (1973) has been able to determine a range of ozone and nitrogen oxide levels capable of causing respiratory illness. This study found that welders continually exposed to 0.01 - 0.36 ppm ozone and 0.01 - 0.08 nitrogen oxides tended to have a higher incidence of chronic lung disease than nonwelders.

ENVIRONMENTAL EFFECTS

ODOR AND VISIBILITY

Studies by Henschler et al, (1960) have shown an odor threshold of below 0.02 ppm for ozone. Results also indicated that the odor diminished rapidly following exposure and was no longer perceptible within a 30 seconds to 12 minute (average 5 minutes) period. The odor persisted longer, an average of 13 minutes, after exposure to an ozone dosage of 0.05 ppm. No experiments have been performed to define an odor threshold for PAN.

Reductions in visibility have been found to be associated with elevated particle concentrations in the atmosphere. High oxidant levels may be important in the production of sulfate or other particles. However, visibility reduction

cannot entirely be explained by oxidant pollution. Denver, Colorado (Russel, 1976, 1977) has haze problems originating from nonphotochemical aerosols. In this case a reduction in oxidant levels would probably not have an effect on reductions of these aerosols and the quantity of haze present in Denver.

LIVESTOCK AND OTHER ANIMALS

No direct effects on domestic animals and wildlife have been found. Laboratory animals studies are discussed in the human health section of this document.

AGRICULTURAL AND OTHER PLANTS

Injury to vegetation was one of the earliest historical manifestations of photochemical oxidant air pollution. Injury from photochemical oxidants has slowed the ripening process and delayed harvest times. This placed an economic burden on the farmers and ranchers whose crops were affected.

In most types of plants, the foliage is the receptor of photochemical pollutants. Oxidants enter foliage through leaf pores called stomata and then dissolve in the water layer inside the leaf, initiating damage in cells surrounding the stomata (tingey, 1977).

The initial effect of oxidants on plants is to increase leakage of water and nutrients from internal cells, causing a decrease in photosynthesis/carbon dioxide fixation and stimulating stress induced ethylene production (Heath, 1975). Subsequently, cellular effects are followed by disruptions in nitrogen fixation and the transfer of metabolites (Gibson, 1976).

Ultimately, biochemical disruptions manifest themselves in three general types of photochemical oxidant plant injury: 1) acute visible injury 2) chronic visible injury 3) physiological effects. (EPA, 1976).

Acute injury usually is visible within 24 hours and is identified by cell collapse with subsequent development of a dead tissue pattern in foliage. It is

associated with short term exposure (hours) at relatively high pollutant concentrations.

Chronic injury, on the other hand, usually is associated with long term or multiple exposure at low pollutant concentration. Chronic injury manifests itself as a progression of tissue discoloration from yellow to brown and terminates with premature foliage drop.

Physiological effects include root alterations, reduced yields, and changes in the quality of plant products. These effects result from either short or long term exposures to photochemical oxidant pollution and may occur without the visible expression of injury.

Ozone

Ozone operates in a cumulative dose-response manner. This means that plants can accumulate injurious doses over long periods of time from relatively low ozone exposures (Table 2). Even at very low concentrations, the period of exposure to oxidants becomes a critical determinant in the severity of pollutant injury. Long-term exposures to low oxidant concentrations eventually may result in substantial negative effects similar in magnitude to certain types of injury caused by higher level acute doses. Different species vary in their sensitivity to ozone, and patterns of ecosystem succession have in several instances changed in economically undesirable directions.

A report prepared by the National Research Council of the National Academy of Science (1976) says that, for an exposure time of one hour, 0.15-0.25 ppm of ozone produces approximately five percent injury, and concentrations of 0.20-0.25 ppm can produce 20 percent injury in sensitive plant species. Five to 20 percent foliar destruction would be economically significant to growers of ornamental plants raised for foliage and flowers.

Forage crops including alfalfa, and root crops such as radish, would be expected to elicit a significant yield loss if 5-20% of their cotyledon leaves

TABLE 2 - OXIDANT - EFFECTS OF LOW CONCENTRATIONS OF OZONE IN
GROWTH, YIELD & FOLIAR INJURY TO SELECTED
MONTANA PLANTS

PLANT	OZONE CONCENTRATION, ug/m ³ (ppm)	EXPOSURE TIME, hours (h)	PLANT RESPONSE AS A REDUCTION FROM CONTROL IN PERCENT (%)	REFERENCES
Alfalfa	98 (0.05) ambient	7hr./day, 68 days	30%, foliage dry wt. harvest 1 50%, foliage dry wt. harvest 2	Neeley <u>et al</u> , 1977 " " " "
Alfalfa	196 (0.10)	2hr./day, 21 days 2hr./day, 21 days	16%, foliage dry wt. 25%, foliage dry wt.	Hoffman <u>et al</u> , 1975 " " " "
Pine, ponderosa	290 (0.15) 290 (0.15)	9hr./day, 10 days 9hr./day, 20 days	4%, photosynthesis 25%, photosynthesis	Miller <u>et al</u> , 1969 " " " "
Pine, ponderosa	290 (0.15) 290 (0.15)	9hr./day, 30 days 9hr./day, 60 days	25%, photosynthesis 34%, photosynthesis	" " " " " " " "
Pine, ponderosa	196 (0.10) ambient	6hr./day, 126 days	12%, root length 21%, stem dry wt. 26%, root dry wt.	Wilhour & Neeley, 1977 " " " " " "
Pine, western	196 (0.10) ambient	6hr./day, 126 days	13%, foliage dry wt. 9%, stem dry wt.	" " " " " "
Wheat, cultivar	392 (0.20)	4hr./day, 7 days (anthesis)	30%, yield	Shannon & Mulchi, 1974
Soybean	98 (0.05)	8hr./day, 5 day/wk. 3 wk. 8 hr./day, 5 day/wk. (mixture of O ₃ & SO ₂ for same periods).	13%, foliar injury 16%, foliar injury 20%, root dry wt.	Timney <u>et al</u> , 1973 " " " " " " " "
Grass, brome	290-647 (0.15)-0.33) (varied)	4hr./day, 5 days/wk. growing season	83%, biomass	Prince & Treshow, 1970 or 1972
Poplar, hybrid	290 (0.15)	8hr./day, 5 days/wk. 6 wk.	50%, shoot dry wt. 56%, leaf dry wt. 47%, root dry wt.	Jensen & Dochinger, 1974 " " "

Table 2 - c 'd.

PLANT SPECIES	OZONE CONCENTRATION, ug/m ³ (ppm)	EXPOSURE TIME, hours (h)	PLANT RESPONSE AS A REDUCTION FROM CONTROL IN PERCENT (%)	REFERENCES
Corn, sweet, cultivar-Golden Midget	98 (0.05) ambient	6hr./day, 64 days	9%, kernel dry wt.	Heagle <u>et al</u> , 1972
Bean, cultivar	255 (0.13)	8hr./day, 28 days	79%, foliage fresh wt. 73%, root fresh wt. 70%, height	Manning <u>et al</u> , 1971 " " " " " " " "
Bean, cultivar	290 (0.15)	2hr./day, 63 days	33%, diomass 46%, pod fresh wt.	Hoffman <u>et al</u> , 1973 " " " "
Bean, cultivar	290 (0.15)	2hr./day, 14 days	8%, leaf dry wt.	Maas <u>et al</u> , 1973
	290 (0.15)	3hr./day, 14 days	8%, leaf dry wt.	" " " "
	290 (0.15)	4hr./day, 14 days	23%, leaf dry wt.	" " " "
	290 (0.15)	6hr./day, 14 days	49%, leaf dry wt.	" " " "
Potatoes 4 cultivars	0.05 0.05	(2 years) 326-533 h	34-50%, yield (2 yr /2cultivars) 20-26%, yield (1 yr /2cultivars)	Heggestad, 1973 " "
Potato cv.	0.15	3 consecutive days	95% injury (leaves withered)	Brasher <u>et al</u> , 1972
Petunia	98-137 (0.0500-0.07)	24hr./day, 53 days	30%, flower fresh wt.	Craker, 1972
8 selected under- story species from an Aspen community	0.15	3hr./day, 5 days/wk. through the growing season	significant amounts of foliage injury and losses in plant and seed wt.	Treshow & Stewart, 1973 " " " " " " " "
Carnation	98-177 (0.05-0.09)	24hr./day, 90 days	50%,flowering (reduced vegeta- tive growth)	Feder, 1970 JUL 27 1970
Geranium	137-196 (0.07-0.10)	9.5/day, 90 days	50%, flowering (shorter flower lasting time, reduced vegetative growth)	Feder, 1970
Poinsettia	196-235 (0.10-0.12)	6hr./day, 5 days/wk. 10 wk.	39%, bract size	Craker & Feder, 1972 " " "

Table 2 cont'd.

PLANT SPECIES	OZONE CONCENTRATION ug/m ³ (ppm)	EXPOSURE TIME, hours (h)	PLANT RESPONSE AS A REDUCTION FROM CONTROL IN PERCENT (%)	REFERENCES
Radish	98 (0.05)	8hr./day, 5 days/wk. 5 wk.	54%, root fresh wt. 20%, leaf fresh wt.	Tingey et al, 1971 " " " "
	98 (0.05)	8hr./day, 5 days/wk. (mixture of O ₃ & SO ₂)	63%, root fresh wt. 22%, root fresh wt.	" " " " " " " "

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(first leaves) were destroyed by oxidant alone or in combination with other air pollutants. Radishes and alfalfa and other plant species, however, have been observed to suffer reduced yields from oxidants even when there was no visible foliar injury.

Table 2 provides experimental results from a number of ozone dose-response experiments performed on plant species growing in Montana either naturally, in agroecosystems, or in home gardens and greenhouses. This table indicates that ponderosa pine, western white pine, alfalfa, corn, white beans, pinto beans, soybeans, hybrid poplar, potatoes, Impatiens, Petunias, Coleus, Geranium, pigweed, Cruciferae genera, aspen, understory plants, and brome grasses, carnation, poinsettia, and radish all can be significantly injured by ozone concentrations of from 0.05-0.15 ppm for various time intervals, and are therefore classified as being sensitive to oxidants.

Foraging livestock, including horses, cattle and calves, sheep and lambs comprise important segments of Montana's agricultural industry. Forage crop damage by oxidants, especially to alfalfa and range grasses such as Bromus would affect maximum yields of livestock. Hay, beans, potatoes, and alfalfa seed are among the 9 most important agricultural crops grown in the state and all are sensitive to oxidant air pollution.

Ponderosa pine is a dominant forest tree species in Montana, and is sensitive to oxidants. Western white pine also is sensitive. Other sensitive Montana plant species important as big game forage and to the stability of natural plant communities include aspen forest understory plants and poplars.

Oxidants affect forests in many ways. One effect is to reduce standing board footage of conifer trees. Montana's number one industry is agriculture, producing some 44 percent of the states total cash receipts with lumbering accounting for an additional 8 percent. Oxidant injury to Montana's timber and agricultural species will have an economic effect on the industries dependent on these species.

PAN

Peroxyacetylnitrate (PAN) as a constituent of the photochemical oxidant mix can cause injury to vegetation. The type of damage caused by PAN is diagnostically different from ozone damage for most acute fumigation episodes (EPA, 1976). The lowest values for the effects of PAN on agricultural plants from data collected from controlled fumigation experiments is: 0.20 ppm for 0.5 hours, 0.10 ppm for 1 hour, and 0.04 ppm for 4 hour exposures (Jacobsen, 1976).

Pollutant Interactions

In the field, vegetation often is injured by a combination of pollutants. Experiments which analyze the injury that would be based upon simultaneous or synchronous fumigations with combinations of pollutants more clearly represent real life situations. Unfortunately, few good studies of this type have been performed.

Creditable studies on ozone and sulfur dioxide generally indicate that these pollutants in combination reduce the effective concentration needed to induce injury either in an additive or greater than additive fashion (Dochinger et al, 1970; Houston, 1974).

Very small amounts of sulfur dioxide and ozone in combination are capable of eliciting damage in vegetation. Chronic exposure to combinations of 0.05 ppm sulfur dioxide and 0.05 ppm ozone produced significant reductions in dry weight content of soybeans and alfalfa (Brennan and Halisky, 1970). These reductions would be particularly significant to Montana's agricultural industry since the concentrations found in these experiments are close to the annual allowable sulfur dioxide standard for Montana and in compliance with the federal 1 hour ozone standard.

Investigations on the effect of PAN and ozone to vegetation have not been definitive. More work in this area is essential.

ECOSYSTEMS

During the last 20 to 25 years, photochemical oxidants have injured both natural and agricultural ecosystems. Oxidants increase the susceptibility of plants to insects and disease. Further, chronic oxidant pollution is linked with crop reductions and a variety of impacts in natural ecosystems (Woodwell, 1970).

In general, oxidant damage to agroecosystems is most dramatic in the short term because such systems are simple, usually consisting of a single plant species with little resilience under pollution. Damage to crops often is immediate and catastrophic. Studies by Johnson (1968, 1970) have shown that oxidant stress reduces photosynthesis and water use by plants, increases leaf and fruit drop, and reduces the marketability of fruit products.

Natural ecosystems, on the other hand, are much more complex and initially more resistant under such stress. Nevertheless, long term oxidant exposure to natural systems disrupts their structure and function and may lead to such effects as soil erosion, a loss of land productivity and many other long term social and economic consequences (Westman, 1970).

Natural ecosystems are structurally complex, relatively stable associations with living and nonliving components interacting in equilibrium. The effects of oxidant pollution on such systems are complex, with most still not completely understood. In general, it is known that disturbances (such as oxidant pollution) return natural ecosystems to a simpler stage in structural and functional development. There is a selective removal of larger overstory vegetation in favor of lower growing forms; a shift from forests toward shrubs and herbs (Woodwell, 1970).

Structural changes in natural ecosystems have been found either directly or indirectly related to oxidant impacts. Wert (1969) found, using aerial photography, that 46,230 acres of a total 160,950 acres of ponderosa-Jeffrey pine forests in the San Bernardino Mountains had severe damage from oxidant pollution. The researchers hypothesized that such damage eventually would result in the

replacement of pollutant sensitive ponderosa and Jeffrey pine by more resistant but less economically and aesthetically desirable trees such as incense cedar. Changes in the tallest, or "canopy" species in a forest eventually results in changes in the lower or "understory" species when the loss or alteration of the taller trees causes changes in the amount of light, water, and other plant growth essentials available to the lower plants (NAS, 1976).

In a series of controlled fumigation experiments, Treshow and Stewart (1973) lent credence to the results found in the San Bernardino studies. They subjected over 70 species indigenous to grassland and aspen-conifer plant communities to 2-hour exposures of 0.15, 0.25, 0.30, and 0.40 ppm ozone. Results indicated that aspen is sensitive to ozone at all those levels.

The researchers considered the sensitivity of aspen to be very significant. Aspen is vital to the aspen-conifer plant community's structural integrity, in that it provides essential shade for young white fir seedlings and other vegetative species. It also was found that an aspen overstory acts as a filter, keeping high ozone concentrations from reaching ground level. Oxidant levels in the San Bernardino Mountains often are greater than the concentrations used by Treshow and Stewart and must be considered capable of causing structural changes in the ponderosa-Jeffrey pine ecosystems located there (Miller et al., 1972).

In altering the vegetation types in natural ecosystems, oxidant pollution indirectly affects all components of the system. Modifications in consumer populations result from disturbances in food availability. Studies (Stark, et al., 1968; Stark and Cobb, 1969) have shown that ponderosa pine weakened by oxidant injury is subject to increased attack by pine bark beetles. It is known from these studies that bark beetles accelerate the changes initiated in the forest community by oxidant exposure.

Such exposure also subjects the decomposer elements of a forest ecosystem to fluctuations. Small animals, arthropods, fungi, and bacteria that depend on forest floor litter as a food source will decrease parallel with a decrease in

the total productivity of the forest ecosystems (Woodwell, 1970).

Small changes brought on by oxidant exposure to natural ecosystems impact and magnify through all trophic levels of the system. As a result of these changes it may take years for the system to reach a new equilibrium, which sometimes may occur only after the replacement of the former system with an entirely different one.

MATERIALS

Ozone and other photochemical oxidants contribute to the degradation of materials. Researchers have identified ozone exposure with accelerated aging in a wide variety of economically important materials, including elastomers (rubber), textile dyes and fibers, and certain paints.

Dose-response data on the affect of ozone on materials are summarized in Table 3. This table indicated the susceptibility range of various materials to ozone. Rubber compounds exposed during the summer appear the most sensitive, showing detrimental effects at levels near natural background. The deterioration rates of paint increased significantly with exposure to 1 ppm ozone. The relatively sensitive paints tested were: oil-based house paint, industrial maintenance paint, latex, coatings on electrical coils, and automotive paint (Sherwin-Williams, 1970). No dose-response data are available for dyes although it has been shown that dyes are sensitive to ozone damage particularly if SO₂ or NO₂ are also present. Garments treated with sensitive dyes have been known to show fading after ten days (Calvin, 1969).

TABLE 3 - MATERIAL RESPONSE TO OZONE

MATERIAL	RESPONSE	EXPOSURE DURATION HR.	CONCENTRATION PPM	REFERENCE
Polybutadiene	Breakage	150-250	0.048 (Summer)	Meyer and Sommer 1957
"	"	400-500	0.042 (Fall)	"
"	"	500-700	0.024 (Winter)	"
Cotton	No Response	1200	0.5 21°C; 72% RH	Morris 1966
Cotton (wet)	Change in fluidity	1200	0.02-0.06	Bogaty <u>et al</u> , 1952
Cotton (dry)	No Response	1200	0.02-0.06	"
House Paint	Increased Deterior- ation	1	1 ppm 66°C 70% RH; followed by 1 hr. darkness 49°C, 100% RH	Campbell <u>et al</u> , 1974
Electrical Coil	"	"	"	"
Coating (Urea-alkyd)	"	"	"	"
Automotive Paint	"	"	"	"
(nitro cellulose/acrylic)	"	"	"	"
Industrial Maintenance Coatings (alkyd)	"	"	"	"

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Measurement Method for Ozone

Measurement - Ref. Method - CFR 40, Part 50, Appendix D.

Ozone present in ambient air undergoes a chemiluminescent reaction with ethylene gas. The emitted light is detected by a photomultiplier and is directly proportional to the ozone concentrations of the ambient air.

The monitors have proven to be highly reliable but it has been difficult to calibrate. These calibration problems are not with the ozone analyzer itself, but instead with the generation of a fixed and stable span concentration.

The original neutral buffered potassium iodide ozone detection reference method was (1) almost confined to the laboratory, (2) unstable over time and (3) very tedious to perform. Gas phase titration and in some cases reverse gas phase titration methods have proved the ozone monitor calibration technique considerably. These methods, however, require the presence of other monitoring devices, eg. oxides of nitrogen analyzers, and well trained personnel.

Recent ozone monitor developments using a ultraviolet light source results in a more stable ozone monitoring device and eliminates the need for the use of ethylene gas cylinders. This rather simple and highly reliable device will probably replace the present chemiluminescence method as present in Appendix D. The EPA has already announced their intentions for this replacement.



CONCLUSION

The EPA (Federal Registrar, June, 22, 1978) has proposed a number of changes in the present oxidant ambient standard. The name of the pollutant would be changed under the proposed EPA revision to ozone, which is the principal measurable ingredient among photochemical oxidants. Furthermore, EPA proposes to change the primary standard to 0.10 ppm on four average from the current 0.08 ppm standard while keeping the secondary welfare-based standard at 0.08 ppm. The review of current literature presented in this paper indicates ozone is the major oxidant capable of scientifically verified adverse health and welfare effects.

Ozone effects in humans appears greatest in portions of the population suffering from asthma and individuals engaged in strenuous exercise. Both these segments of the public demonstrate adverse effects on lung function near the proposed 0.10 ppm standard. Whether this standard is adequate to protect these individuals hinges on the definition of what is an adequate margin of safety.

The secondary welfare standard (protection of materials, ecosystems, and plant species) of 0.08 ppm proposed by EPA is not adequate to protect all sensitive plant species within Montana. This conclusion is based on the experiments presented in the section on agriculture and upon an analysis of Montana's ambient air quality data for ozone.



Other State Standards

PHOTOCHEMICAL

ALABAMA - 1 hr. 0.1 ppm (200 ug/m³)

ALASKA - 1 hr. 160 micrograms

ARIZONA - 1 hr. 160 micrograms

CALIFORNIA - 0.10 ppm 1 hr.

CONNECTICUT - 160 micrograms (0.08 ppm) 1 hr.

COLORADO - 0.1 ppm^a 0.2 mg/M³^b

DELAWARE - 1 hr. (0.08) 160 micrograms

FLORIDA - 160 (0.08 ppm) max 1 hr.

GEORGIA - 98 micrograms 1 hr. 25°C and 760 mm.Hg.

HAWAII - 98 micrograms 1 hr. 25°C and 760 mm.Hg.

IDAHO - 160 micrograms (0.08 ppm) 1 hr.

ILLINOIS - .080 ppm 1 hr.

INDIANA - 160 ug/m³ (0.08 ppm) 1 hr. (O₃) 1,965 ug/m³ = 1.0 ppm

IOWA - none

KENTUCKY O₃ 160 micrograms 1 hr. (0.08 ppm)

LOUISIANA - 160 ug/m³ 0.08 ppm max 1 hr.

MAINE - 1 hr. 160 micrograms

MARYLAND - ug/m³ 160 ppm ozone 0.08 ug/m³ 1960= ppm

MASSACHUSETTS - 10 milligrams (9 ppm) 8 hr. 40 milligrams 35 ppm 1 hr.

MICHIGAN - (O₃) 0.6 ppm (1200 ug/m³ for any hour

MINNESOTA - 0.07 ppm 130 micrograms 1 hr.

MISSOURI - 0.10 ppm 1 hr.

MONTANA - 160 ug/m³ 0.08 ppm 1 hr.

NEW HAMPSHIRE - 160 ug/m³ 0.08 ppm

NEW JERSEY - 1 hr. 160 micrograms 0.08 ppm

NEW MEXICO - 1 hr. 0.06 ppm

NORTH CAROLINA - 160 0.08 ppm 1 hr.

NEW YORK - 1 hr. 0.80 Max.

NORTH DAKOTA - 160 0.08 ppm 1 hr.

OHIO - 1 hr. 119 micrograms 0.06 ppm

4 hr. 79 micrograms 0.04 ppm

24=hr. 40 micrograms 0.02 ppm

OREGON - 160 micrograms 0.08 ppm 1 hr. average

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SOUTH DAKOTA - 160 1 hr.

PENNSYLVANIA - National Standards

VIRGINIA - 160 micrograms 0.08 ppm 1 hr.

WASHINGTON - 1 hr. 160 $\mu\text{g}/\text{m}^3$ 0.08 ppm. 10-4p.m. April-1-Oct 31

WEST VIRGINIA - 1 hr. 160 0.08 ppm

SOUTH CAROLINA - 1 hr. 160 $\mu\text{g}/\text{m}^3$ 1 hr, 100

TENNESSEE-160 $\mu\text{g}/\text{m}^3$ 0.08 ppm 1 hr. avg.

UTAH - .08 ppm 1 hr.

VERMONT - 118 $\mu\text{g}/\text{m}^3$ 0.06 ppm

WASHINGTON D.C.-160 $\mu\text{g}/\text{m}^3$ 1 hr. 0.08 ppm
160 $\mu\text{g}/\text{m}^3$ 1 hr. 0.08 ppm

WISCONSIN - 160 micrograms.08 ppm 1 hr.

WYOMING - 160 micrograms 0.08 ppm 1 hr.

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Chapter II

HYDROCARBONS

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Introduction and Summary

Hydrocarbons are chemical compounds with a molecular structure of carbon and hydrogen. Some hydrocarbons are thought to be relatively harmless by themselves, but may react with other air pollutants and sunlight to form photochemical oxidants which may be harmful.

The hydrocarbons which cause most of the air pollution are relatively low molecular weight compounds produced by the incomplete combustion of fuels and refuse and by the evaporation of volatile compounds. In Montana (and the nation as a whole) automobiles and other transportation sources contribute the majority of the hydrocarbons. Pollution control devices and engineering improvements have substantially reduced the concentrations of hydrocarbons emitted by the automobiles since the mid 1960s. In most urban areas, however, hydrocarbons from automobiles are still a major air pollution problem.



Existing Hydrocarbon Pollution

Sources and Emissions

The primary sources of hydrocarbons are the incomplete combustion of fuel in internal combustion engines and evaporative gasoline emissions. Exhaust hydrocarbons have been reduced by approximately 90% since 1968, due to engine modifications and pollution control devices on the newer model automobiles. Evaporative gasoline emissions have been reduced by 30% since 1971 by pollution control devices. Even though hydrocarbons from conventional automobiles have been reduced significantly since the mid 1960s, they are still the major source of photochemically reactive hydrocarbons in urban areas. It has been estimated that for a typical 1975 model car 60% of the hydrocarbons lost are emitted as evaporative gasoline losses from the carburetor and fuel tank with the remaining 40% lost through the exhaust (EPA, 1976).

Diesel engines produce less hydrocarbon pollution because they burn fuel more completely and have negligible evaporative loss. Jet aircraft engines produce hydrocarbon pollution in the exhaust but in relatively small quantities.

Natural sources of hydrocarbons include: biological decomposition of organic materials, range and forest fires, and vegetation. Although relatively large amounts of methane are produced by biological decomposition, this hydrocarbon is photochemically inactive and therefore of little concern as a pollutant.

Data for hydrocarbon emissions in Montana (Ewing, 1971), shows that the greatest amounts come from motor vehicles and other transportation sources. Of a total hydrocarbon production estimate for Montana (182,752 tons per year) approximately 50% (90,898 tons per year) came from motor vehicles. The following table further breaks down the amounts of total hydrocarbons from various sources:



Source	Tons of Hydrocarbon per year	% of Total
Fuel combustion (coal, gas, oil)	3,093	1.7
Process losses (grain elevators, asphalt batching) (plants, etc)	43,447	23.8
Solid waste disposal (open burning, slash burning)	28,966	15.8
Transportation (motor vehicles,) (aircraft, railroads)	90,898 16,384	49.7 9.0
Total	182,752	100

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Ambient air quality data for hydrocarbons in Billings Montana shows that the total hydrocarbon values are high. Because data is not available, on the relative amounts of methane and non-methane hydrocarbons, it is not possible to determine if there is a significant air quality problem. The federal guide line for non-methane hydrocarbon is .24 ppm between 6 and 9 a.m.

If we subtract an estimated methane hydrocarbon concentrations of 6 ppm from the measured total hydrocarbon values, estimated concentrations of non-methane hydrocarbons can be computed. The highest estimated average value for nonmethane hydrocarbons between 6:00 and 9:00 a.m. was 21.7. If the estimate of 6 ppm for methane hydrocarbons in Billings is close to the actual value, the suggested 3 hour average concentration exceeds by approximately 100 times the federal guideline level.

Human Health Effects

Under certain atmospheric conditions hydrocarbons react to form photochemical oxidants as has been previously discussed. Some of these reactive hydrocarbons and others that do not participate in the photochemical reaction

may also have adverse health effects of their own. The aromatic hydrocarbons cause blood and nervous system disorders as well as irritation to the membranes of the eye and respiratory tract. There is also evidence that benzene and other hydrocarbon cause cancer. The aldehydes of most concern in air pollution are formaldehyde and acrolein. They contribute to the odor and eye irritation often associated with urban air pollution. Because very little research has been done at ambient levels, little is known about effects at low-level exposures.

Environmental Effects

Odor and Visibility

Hydrocarbons in the urban environment contribute to odor and visibility problems by reacting photochemically to produce oxidants. These oxidants are the main ingredients of the dense and odorous urban "smog".

Some hydrocarbons have no detectable odor whereas others, particularly the aromatics and aldehydes, produce detectable odor. Among these, acrolein and formaldehyde concentrations have been shown to correlate with the intense odor of diesel exhaust (Elliott and Davis, 1950).

Agriculture and other plants

Among hydrocarbons, ethylene has the greatest potential for adverse impact on agricultural and native plant communities. It is a plant growth regulator under natural conditions and is harmful to plants at levels higher than those found in an unpolluted environment. Experimental concentrations of ethylene as low as 0.005 ppm (for 24 hours) may have a harmful effect on plant growth and development. Agricultural crops such as orchids, cotton, squash, beans and peas are sensitive to ethylene damage. Based on economic data from California, annual losses suffered by carnation and orchid growers from ethylene damage were estimated in excess of \$7,000,000 per year (James, 1963).

Measurement Methods for Hydrocarbons

Reference method - CFR 40, Part 50, Appendix E. A hydrogen flame ionization detector is used to measure total hydrocarbons while a stripper column within the gas chromatograph removes moisture, carbon dioxide and non-methane hydrocarbons.

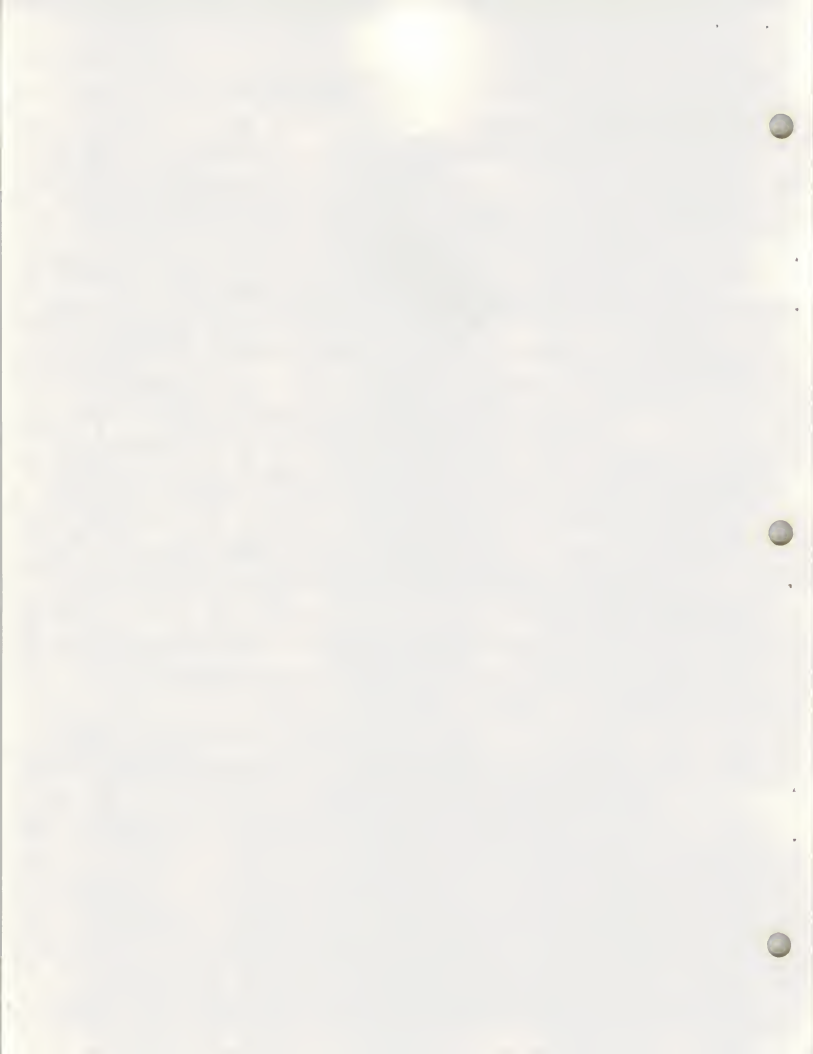
This reference method has some limitations as follows:

- (1) The gas chromatograph is calibrated usually for one or two specific gases (e.g., methane and propane) but it is called upon to measure many different hydrocarbon concentrations within urban atmospheres. The device responds to the numbers of carbon atoms within gaseous hydrocarbon compounds.

If it is calibrated for low molecular weight hydrocarbons, it measures these smaller compounds more accurately than it does the compounds with high carbon counts. If the device were calibrated for large carbon compounds, it would measure them more accurately than the small compounds. It is difficult, therefore, to characterize the entire gaseous hydrocarbon using any given hydrocarbon analyzer with a given separation column.

Methane is ubiquitous at concentration levels of about 1.5 parts per million. Because of this characteristic level, the reference method corrects hydrocarbon concentrations for methane.

- (2) Gas chromatographs are laboratory devices. Their use in the field is difficult and generally requires highly competent and trained personnel for correct operation.



Other State Standards

HYDROCARBONS

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ARIZONA - 6-9 a.m. 160 micrograms

CONNECTICUT - 160 micrograms (0.24 ppm) max 3-hr. 6-9 a.m.

DELAWARE - 3 hr. 2/10 0.2 - 131 micrograms

FLORIDA - 160 (0.24 ppm) 3 hr. 6-9 a.m.

GEORGIA - 98 micrograms, 3 hr. 25 degrees C and 76 mm Hg

HAWAII - 3 hr. 100 micrograms

IDAHO - 160 micrograms (0.24 ppm) 3-hr. 6-9 a.m.

ILLINOIS - 6-9 a.m. 3-hr. .24 ppm

INDIANA - 160 ug/m³ (0.24 ppm) 3-hr 6-9 a.m. (CH₄)

KENTUCKY - 160 micrograms (0.24 ppm) 3-hr. 6-9 a.m.

LOUISIANA - 160 ug/m³ (0.24 ppm) 3-hr.

MAINE - 3-hr. 160 micrograms

MARYLAND - 3-hr. 6-9 a.m. $\frac{\text{ug/m}^3}{10}$ $\frac{\text{ppm carbon}}{0.24}$ $\frac{\text{ug/m}^3}{655} = \text{ppm}$

MASSACHUSETTS - 3 hr. 6-9 a.m. 160 ug/m³ 0.24 ppm

MINNESOTA - 0.24 ppm 160 micrograms 3-hr. 6-9 a.m.

MONTANA - 160 ug/m³ 0.24 ppm 3-hr. 6-9 a.m.

NEVADA - 3-hr. 6-9 a.m. 160 ug/m³ 0.24 ppm

NEW HAMPSHIRE - ug/m³ 6-9 a.m. 160 ug/m³

NEW JERSEY - 6-9 a.m. 160 micrograms (0.24 ppm)

NEW MEXICO - 3-hr. 0.19 ppm

NORTH CAROLINA - 160 micrograms (0.24 ppm) 3-hr. 6-9 a.m.

NEW YORK - 3-hr. 6-9 a.m. 0.24

NORTH DAKOTA - 160 micrograms (0.24 ppm) 3-hr. 6-9 a.m.

OHIO - 3-hr. (126) micrograms (0.19 per million) 6-9 a.m.

OREGON - 160 micrograms (0.24 ppm) 3-hr. 0600 to 0900

SOUTH DAKOTA - 160 micrograms 3-hr.

UTAH - National Standards .24 ppm 3-hr. 6-9 a.m.

Hydrocarbons cont'd

WASHINGTON - 3-hr. 6-9 a.m. 160 ug/m³ 0.24 ppm

VIRGINIA - 160 (0.24 ppm) 3 hr.

WEST VIRGINIA - 8 hr. 10 mill (9 ppm) 1 hr, 40 mill (9 ppm)

WASHINGTON D.C. DISTRICT OF COLUMBIA - 160 ug/m³ (3 hr) 0.24 ppm 6-9 a.m.

WISCONSIN - 160 (.24 ppm) 3-hr. 6-9 a.m.

WYOMING - 160 (0.24 ppm) 3-hr. 6-9 a.m.

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NITROGEN OXIDES

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I. Introduction

Nitrogen oxides are associated with fossil fuel transportation and other combustion processes. Among the oxides of nitrogen, nitrogen dioxide is highly toxic in small quantities, and therefore causes the most concern. Most of the emitted gas is nitric oxide which is subsequently converted to nitrogen dioxide in the atmosphere.

Nitric oxide and nitrogen dioxide are emitted from man-made sources. Other oxides of nitrogen are formed from photochemical reactions (chemical conversions caused by sunlight) in the atmosphere and may contribute to acid rain (Whitten and Hugo, no date).

The nitrogen oxides may react with hydrocarbons to form photochemical smog. Photochemical smog is composed of a variety of oxidants harmful to human health and the environment. Evidence indicates that photochemical oxidants, nitrogen oxides and sulfur oxides, may all act together in causing human health effects. An overview of the transportation pollutants and the section on ozone and PAN will discuss these interrelationships.

Exposure to nitrogen dioxide for short periods may cause harmful reactions to sensitive individuals, such as persons with asthma or other respiratory disease.

The long term effects are more effectively determined in epidemiological studies indicating increased respiratory infections and related impairments of the respiratory system. These effects actually may result from a series of short term exposures to nitrogen dioxide. Correlations between nitrogen oxide exposures and increased cancer have been reported, but nitrogen oxides have not definitely been established as the cause.

Ambient air quality standards should reflect an understanding of nitrogen dioxide effects for both short term exposure and the cumulative effects of repeated



exposures. The standards also should take into consideration the hydrocarbons and oxidants involved with nitrogen oxides in photochemical processes, with possible additive effects on human health.

II. Nitrogen Oxide Pollution In Montana

A. Sources and Emission Data

Most of the oxide of nitrogen in the urban atmosphere originates from the combustion of fossil fuels, both in Montana and the rest of the nation. Approximately one-half of the nitrogen oxides are from the transportation sources while the others are primarily from industrial fuel combustion (EPA, 1975).

The Montana data for all sites are listed in Table I (Ewing, et al., 1971). Slash burning and teepee burners contributed much higher percentages of the total in the Missoula area than in the nation as a whole.

B. Ambient Data

Ambient air data for the major metropolitan areas in the United States indicate that areas with high concentrations of photochemical smog have relatively high peak concentrations of nitrogen dioxide. Maximum peak values for one hour concentrations of nitrogen dioxide between 500-1200 ug per cubic meter were common for large cities because of photochemical reactions. The peak concentrations were approximately 5 to 15 times the annual average concentrations for the pollutant (EPA, 1977).

The available data for Montana recorded near traffic centers and other urban areas are listed on Table II for annual average concentrations. Montana has lower concentrations than those found in the larger urban areas of the country.

The highest one hour concentration was .18 ppm and occurred at the Billings 27th and Montana Street sampling site on August 2, 1977 at 9 A.M. The yearly average for 1977 (recorded through September only) was approximately .03 ppm.

Since the current Montana and Federal Standard is .05 ppm NO₂ as a 1-year average, the ratio of peak concentration to annual concentrations should reflect the relative protection a 1-hour standard will provide compared to a 1-year average.

III. Health Effects

A. Community Studies

Chronic exposure studies usually have been epidemiological or conducted with experimental animals.

The current federal ambient air standard is based on a chronic exposure period of one year. The isolation of nitrogen dioxide concentrations as a cause of health effects is difficult because exposures occur over long periods, and because other pollutants, climatic differences, and variable population make up influence the results. Further atmospheric photochemical reactions convert the nitrogen oxides into different compounds which may cause the effects.

One of the more significant epidemiological studies involved the Chattanooga area in Tennessee. Epidemiological evidence indicated that children suffered breathing difficulty. An 18 percent increase in respiratory disease also was observed. Acute bronchitis was noted in areas with elevated nitrogen dioxide. Levels of average exposure were between 118 and 156 ug/M³ (.063 and .083 ppm). This study was heavily relied upon as justification for the current federal standard of .05 ppm, (Shy, C.M., 1970); (Shy et al., 1970). These studies have been criticized because of the unreliable ambient air measurement used. Pearlman et al., (1971) studied the same area again and found a correlation between length of time of exposure and frequency of bronchitis. Chapman et al., (1973) studied chronic bronchitis in the parents of high school students in Chattanooga. They did not, however, find a correlation between the different ambient concentrations recorded during the study and the prevalence of chronic respiratory disease.

Investigations of the effect of nitrogen dioxide in houses where gas stoves

cause excessive nitrogen dioxide concentrations support the conclusion that low levels (.5 ppm range) of nitrogen dioxide increase respiratory illness in children (Melia et al., 1977; Wade et al., 1975). These findings were not repeated in a study in Ohio by Lutz et al., (1974). However, a much smaller sample size was used in these investigations.

Studies in the Soviet Union and Czechoslovakia have indicated other respiratory disease problems associated with individuals living near nitrogen dioxide sources. These studies indicated harmful effects from average nitrogen dioxide concentrations of less than .10 ppm. One study indicated average levels between .02-.07 ppm caused enlargement of lymph nodes and tonsils and changes in blood characteristics. These studies have not been reproduced or their results confirmed by other investigators (NAS, 1973).

Excessive mortality rates caused by various cancers and heart and respiratory disease as related to air pollution have been studied by Hickey et al., (1970). Exposure to nitrogen dioxide, sulfur dioxide, cadmium, and other pollutants has been correlated with increases in lung and breast cancer and arterial disease. The lack of experimental evidence supporting these correlations does not allow any conclusion, but points to the need for further study.

B. Controlled Exposure Studies

Recent speculation on previous epidemiological studies indicates that what appeared to be long term chronic effects may instead have been the accumulation of short term acute effects. Table III summarizes the important health effects measured in key experiments.

The senses are affected by lower concentrations of nitrogen dioxide than any other physiological system.

The threshold for odor perception of nitrogen dioxide is approximately .11 ppm. The odor will not be noticed after a short length of time because nitrogen

dioxide affects odor perception by dulling the sensors. Gradual increases of up to 27 ppm were made before this impairment was overcome and the odor was again detected (Feldman, 1974).

An additional effect is the interference with the dark adaption reflex. Very low concentrations of nitrogen dioxide increase the length of time it takes to adapt to darkness. Concentrations of .07 ppm to .26 ppm have been reported to produce significant impairment of dark adaption (Shamberidze, 1967; Bondareva, 1963).

Effects on pulmonary function have been recorded above 1 ppm. The lowest concentration producing any measurable effect was 1.1 ppm which increased sensitivity of the air passages to a drug used experimentally to induce the early stages of asthma attacks in asthmatic persons (Orehek., 1976).

Current efforts in establishing a nitrogen dioxide standard have primarily been concerned with the effects of nitrogen dioxide and generally have neglected the effects of the products resulting from the photochemical oxidant reaction sequence associated with nitrogen oxides. As described in the photochemical oxidant section, nitrogen oxides play an important part in the formation of these oxidants which have their own effects on human health. Health effects of the oxidant products are described in the photochemical oxidant report.

C. Animal Studies

Studies with experimental animals are important because the effects of long term-low level nitrogen dioxide exposures on lung tissue can be studied in a controlled environment along with variable levels of other pollutants found in the ambient environment of humans. Very low levels of NO₂ have caused significant adverse effects by increasing susceptibility to infectious lung disease.

Blair et al., (1969) reported that concentrations of .5 ppm for 6 to 24 hours per day over three weeks produced emphysematous conditions in experimental

mice. Ehrlich and Henry (1968) reported that .5 ppm produced increased susceptibility to bacterial pneumonia in mice exposed for three continuous months. Many other effects have been reported in experimental animals at higher doses, usually in excess of 1 ppm.

In guinea pigs, short term exposure of .5 ppm of nitrogen dioxide produced protein in the urine, indicating a kidney disorder (Sherwin and Layfield, 1974). Other studies are included in Table III.

IV. Environmental Effects

A. Odors and Visibility

Oxides of nitrogen react in the presence of sunlight to form atmospheric oxidants. The oxides of nitrogen and photochemical oxidants vary in their influence on visibility and the sense of smell. Nitric oxide is a colorless, odorless gas whereas nitrogen dioxide is a reddish-orange-brown gas with a pungent odor. Nitrogen dioxide in the atmosphere causes a reduction in light by absorbing sunlight of certain wave lengths. Nitrogen dioxide and particulate matter combine to reduce the visibility, contrast and brightness of distant objects (EPA, 1971).

Nitrogen dioxide odor is discernible at very low concentrations. However, it dulls the senses and decreases the ability to determine nitrogen dioxide odors. Adaptation to the gas is relatively quick and odors are not detected in normal environmental situations unless rapid changes in concentrations occur. Odor detection is described in more detail in the Health Effects section.

B. Agriculture and Other Plants

Damage to vegetation resulting from direct exposure to nitrogen oxides in the ambient air usually has been observed both in the proximity of industrial sources and as a result of experimental exposure (EPA, 1971). Thomas (1951) reported on damage to vegetation in the vicinity of a nitric acid plant as a result of escaping nitric oxide gases. Cotton, pinto beans and endive plants, experimentally exposed to 1.0 ppm nitrogen dioxide for 48 hours, showed slight

leaf damage. Concentration of 3.5 ppm for 21 hours completely killed pinto beans and endive and moderately damaged cotton (Heck, 1964). Species of citrus fruits, tobacco and soybean have also been exposed to experimental concentrations and damage has been observed. At nitrogen dioxide concentrations of 0.25 ppm for an 8-month period, leaf-drop was observed in navel oranges (Thompson, 1970).

Experimental exposure of agricultural plants has shown that some are more sensitive than others to nitrogen dioxide. The threshold for plant injury, defined as the exposure required to injure five percent of the leaves, has been tabulated (EPA, 1971) for a number of plants. Table IV presents the data.

There are a few literature references regarding the effects of nitrogen dioxide on range land and native species of vegetation. Hill et al., (1974) exposed native vegetation in Arizona to a mixture of sulfur dioxide and nitrogen dioxide at concentrations of .05-11 ppm sulfur dioxide and 0.1-5 ppm nitrogen dioxide. The authors observed no damage.

C. Livestock and Other Animals

There appear to be no data in the literature on the effects of nitrogen dioxide on livestock and wildlife. Laboratory animals have, however, been dosed with nitrogen dioxide and the effects have been recorded.

Death from exposure to nitrogen dioxide in experimental animals is usually caused by fluid accumulation in the lungs, oxygen deprivation, and inflammation of lung tissue.

Effects on experimental animals are discussed in the Human Health Section.

D. Ecosystems

Generally, most adverse impacts of oxides of nitrogen on the environment appear to be related to the role nitric oxide and nitrogen dioxide play with hydrocarbons and sunlight in the formation of photochemical oxidants. Photochemical oxidants are biologically and chemically active compounds which have adverse impacts on health, vegetation, materials, visibility and smell.

A more complete discussion of the role nitrogen dioxide and other oxides of nitrogen play in the photochemical sequence is included in the section of this report on photochemical oxidants.

E. Materials

Nitrogen oxides have been known for some time to fade certain dyes. Most of the investigations of this phenomena have established the lowest concentrations causing such damage. Concentrations of .6 ppm in home clothes dryers operated by natural gas have been shown to cause fading in fabrics (EPA, 1971).

A corrosive effect in nickle-brass alloys has been observed on various components of the telephone system in California urban areas. The corrosion usually is related to nitrate particulates that have been deposited on sensitive relays or other areas. This type of nitrate particulate accumulation has been associated with an average concentration of nitrogen dioxide as low as .066 ppm (EPA, 1971).

V. Conclusions

The federal government is currently proposing a short term standard based on the human health effects of exposure to concentrations of nitrogen dioxide for less than one hour. Available data indicate that bronchial effects on asthmatics occur at very low concentrations, but it is not generally agreed that the effects can be considered adverse. Sensory effects of very low levels likewise are not necessarily considered adverse. Changes in lung tissue and susceptibility to respiratory disease and other physiological impacts have been noted. The concentrations that produce these effects are in the .5 ppm range.

Besides causing impacts by itself, nitrogen dioxide can be a precursor for oxidant formation at very low concentrations (less than .05 ppm). Effects of the oxidants are addressed in the oxidant portion of this working paper.

Annual averages of nitrogen dioxide of approximately .06 ppm have been associated with material damages. Further, correlation studies of community

TABLE I- SOURCE CATEGORY NITROGEN DIOXIDE EMISSIONS PROJECTS
(Table values are in tons of pollutants/year-1970)

SOURCE:	Great Falls	Helena	Missoula	Billings	Miles City
I. Fuel Combustion	1,816	2,623	1,464	9,547	5,694
II. Industrial Process Losses	36	--0--	260	373	--0--
III. Solid Waste Disposal					
A. Open burning	370	267	344	172	202
B. Slash burning teepee burners	89	1,322	9,715	291	6
IV. Transportation-Area Sources	13,828	15,310	15,347	13,825	10,227
Miscellaneous Sources					
A. Agricultural burning	52	25	8	27	62
B. Forest wildfires	703	2,243	4,178	467	126
TOTAL FROM ALL SOURCES	16,394	21,790	31,316	24,702	16,317

Reference: Ewing, et al. , 1971.

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exposures have related annual average concentrations of less than .10 ppm to increased bronchitis in children. These may be cumulative effects of short term exposures to peak concentrations which are much above the annual average.

Examination of current ambient data in Montana indicates that an ambient air standard of one hour concentrations not to exceed .25 ppm would provide the same protection as the current .05 annual average standard. This standard would not be adequate to protect against sensory effects or to prevent nitrogen dioxide levels of high enough to act as an oxidant precursor in the formation of ozone concentrations.

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TABLE II Maximum One-Hour Average Concentrations and Annual Average Concentrations Recorded for Nitrogen Dioxide at Various Montana Locations.
(Table values listed in ppm)

DATES	LOCATIONS					
	Billings*		Missoula		BUTTE	
	1 hr.	Annual Avg.	1 hr.	Annual Avg.	1 hr.	Annual Avg.
1/78	.10	.039				
11/77			.09	.03		
9/77	.12	.029				
8/77	.18	.029			.06	.012
7/77	.14	.018			.06	.015
6/77	.08	.012				
5/77	.06	.01				
4/77	---	---				
3/77	---	---			.08	.022
2/77	.04	.023				
8/76	.08	.032				
7/76	.11	.033				
6/76	.06	.034				
5/76	.07	.036				
4/76	.06	.039				
3/76	.10	.041				
2/76	.08	.043				
1.76	.10	.048				
12/75	.10	.041				
11/75	.05	.030				

* Billings monitor located at 27th and Montana; Missoula at Lion's Park; Butte at Alpine West.
AQB monitoring data.

TABLE III Effects on Humans and Animals of Short Term Exposures to NO₂.

NO ₂ Concentrations ug/m ³ ppm		Human Studies		NO ₂	Measurement Method
		Community Studies	Controlled Exposure Studies	Animal Studies*	
3,760	2.0			A single 3 hr exposure caused increased mortality following challenge with an infectious agent. (Ehrlich <u>et al.</u> , 1977).	Chemiluminescence
3,000	1.6		Exposure for 15 min caused significant increase in R _{aw} in patients with chronic bronchitis. (von Nieding and Krekeler, 1971).		Saltzman
2,800	1.5			After 1 wk. continuous exposure, mice had a significantly greater increase in susceptibility to infectious pulmonary disease compared to intermittent exposure.	Saltzman procedure used in human studies. Saltzman and Chemiluminescence procedures used in animal studies.
188 & daily 2 hr spike 1,880	0.1 continuous & daily 2 hr spike 1.0			Emphysematous alterations in mice after 6 month exposure. (Port <u>et al.</u> , 1977).	Saltzman

TABLE III(continued)

NO ₂ Concentrations, ug/m ³ ppm		Human Studies		NO ₂ Measurement Method
		Community Studies	Controlled Exposure Studies	
940	0.5			Animal Studies* In mice challenged with bacteria, there was increased mortality following a 90 day continuous exposure or a 180 day intermittent exposure. (Ehrlich and Henry, 1968).
940	0.5			Animal Studies* A 7 day-intermittent exposure caused enzymatic alterations in lungs and blood. (Donovan <u>et al.</u> , 1976). Intermittent exposure for 6 hr/day for up to 12 mo. caused morphological changes. (Blair <u>et al.</u> , 1969).
190	0.1		Exposure for 1 hr reported to cause increased R _{aw} in some asthmatics, who were also exposed to a broncho-constrictor which increased susceptibility to NO ₂ . Statistical treatment of data raises questions as to validity of results. (Orehek <u>et al.</u> , 1976)	Saltzman

TABLE III(continued)

NO ₂ Concentrations		Human Studies		Animal Studies	NO ₂ Measurement Method
ug/m ³	ppm	Community Studies	Controlled Exposure Studies		
470	0.25	This range of concentration of NO ₂ has been shown to occur regularly in the vicinity of gas stoves during meal preparation. Highest recorded value was 1,880 ug/m ³ (1.0 ppm). A study of children living in homes in which gas stoves were used showed higher rates of respiratory symptoms and disease when compared to children living in homes in which electric stoves were used. (Melia <u>et al.</u> , 1977; Wade et al., 1975).			
to	to				
940	0.5				

NO₂ 1.2 10³
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TABLE IV - Plant Threshold Susceptibility To Acute Injury
From NO₂

<u>Species</u>	<u>Threshold Concentration (ppm)</u>	<u>Time (hr)</u>
endive	1.0	48
lettuce	4	1.5
mustard	20	4
oats	4	1
wheat	1	7
alfalfa	30	1
bean	10	4
cotton	1	12
navel oranges	1	35
tomato	8	1
tobacco	2.4	5
pepper	5.5	1.5

Nitrogen Dioxide Measurement Method

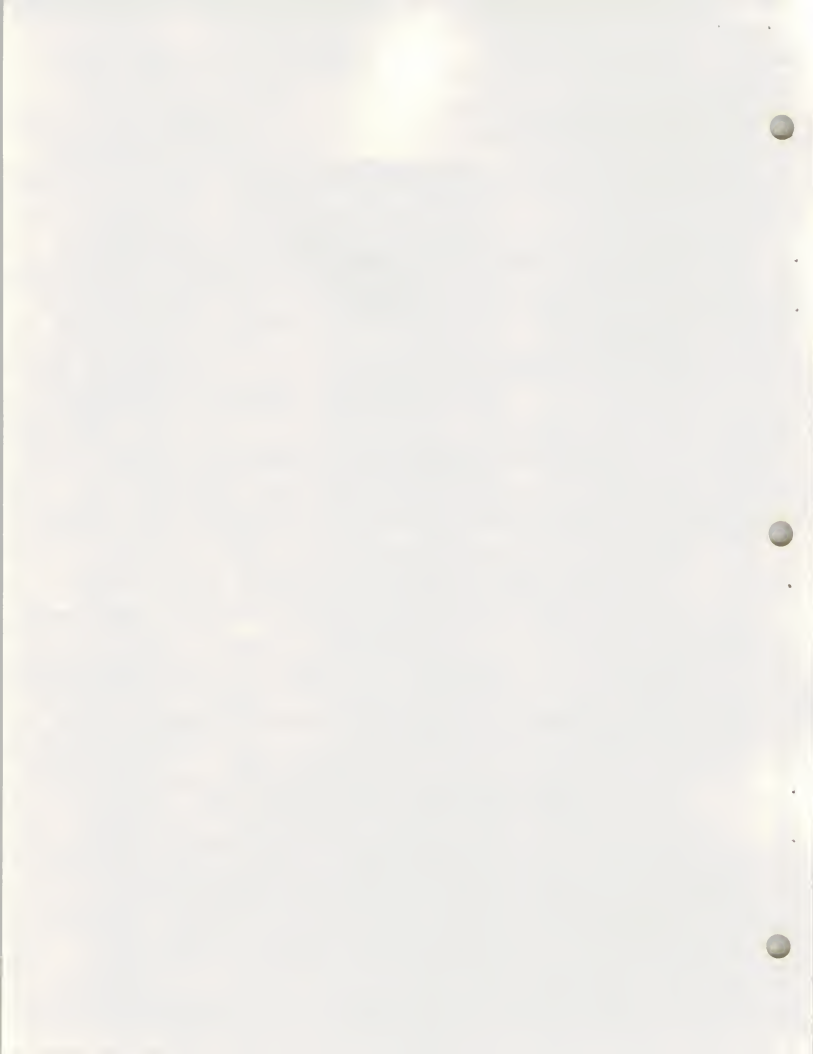
Measurement - Reference Method - CFR 40, Part 50, Appendix F.

Nitrogen dioxide is measured indirectly by an electronic (or mathematical) subtraction of two other directly measured compounds. Nitric oxide is measured by a flameless photon-emitting reaction (with ozone). A measure of total oxides of nitrogen is determined by converting all non-nitric oxide compounds to nitric oxide, and then determining this concentration in the same manner as nitric oxide. Nitrogen dioxide is the difference between the total oxides of nitrogen and the nitric oxide concentration.

The non-nitric oxide compounds (mostly nitrogen dioxide) are reduced to nitric oxide by means of a catalytic converter. The difference between the readings obtained through the converter and the readings obtained without going through the converter is the nitrogen dioxide concentration.

Nitric oxide and ozone react very quickly and emit a photon during this reaction. The photon is detected by a photomultiplier tube and amplified to the desired signal.

All nitrogen dioxide analyzers are quite complicated both in their plumbing scheme and electronics. All require precise controls over timing mechanisms and electronic amplification. Some single detector analyzers can show temporary false positive and negative values during parts of the cycle if the concentrations of oxides of nitrogen are changing. Other dual detector analyzers require extended care in balancing of electronic amplifier signals and air flow velocity through the analyzer. Calibration of the nitrogen dioxide analyzer requires a careful and precise technique.



Other State Standards

NITROGEN OXIDES

ALABAMA - 24-hr. 0.15 ppm (282 ug/m³ 1 hr. 0.6 ppm (1130 ug/m³)

COLORADO - 0.20 2 hr.

DISTRICT COLUMBIA - 0.20 2 hr.

HAWAII - 150 micro 24 hr.

INDIANA - 100 ug/m³ (0.05 ppm) 1,880 ug/m³-1.0 ppm.

KENTUCKY - 100 micrograms (0.5 ppm)

MINNESOTA - 0.05 ppm 100 micrograms

MONTANA - 100 ug/m³ 0.05 ppm annual

NEW HAMPSHIRE - 0.05 ppm 100 ug/m³

NEW JERSEY - 12 months 100 micrograms (0.05 ppm)

NORTH CAROLINA - (0.05 ppm) 100 micrograms

OREGON - (0.05ppm) 100 micrograms

PENNSYLVANIA - National Standards

SOUTH CAROLINA - 100 micrograms annual

SOUTH DAKOTA - 100 micrograms, 250 micrograms 24 hr.

UTAH - .05 ppm National Standards.

VIRGINIA - 100 micrograms (0.05 ppm) annual arith mean

WISCONSIN - 100 (0.05 ppm)

WYOMING - 100 micrograms (0.05 ppm)

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CARBON MONOXIDE

Introduction

This brief review of carbon monoxide (CO) concentrations on the effects produced by ambient air concentrations in the Montana environment.

Carbon monoxide at low concentrations contributes to human health problems. Therefore, air quality standards are aimed at protection of human health. Very small increases in background carbon monoxide have been shown to produce a decrease in human performance and adverse effects in individuals with certain types of cardiovascular disease.

Effects of ambient carbon monoxide appear to be minimal on vegetation, materials, and aesthetics.

High ambient concentrations of carbon monoxide recorded in Montana appear to be related to transportation, particularly in busy urban areas.

Eight hour exposure to concentrations of nine ppm (the current federal standard) should produce carboxyhemoglobin in the human blood stream at concentrations that have been correlated with impaired aerobic performance and adverse effects on individuals suffering from cardiovascular disease.

Carbon monoxide effects also are dependent on the amount of oxygen in the atmosphere. A given concentration of carbon monoxide will have a greater effect on an individual if the oxygen concentration in the atmosphere is reduced. Because of this relationship, an ambient standard based on sea level concentrations of oxygen would not provide the same amount of protection at higher elevations. At 5000 feet (the approximate altitude of Butte) the federal standard would allow, on a relative basis, 22 percent more carbon monoxide than at sea level. Thus, a standard of 8.7 ppm would result in physiological effects equal to those from an exposure at 10.3 ppm (NAS, 1974).

A definite lower threshold for adverse effects caused by carbon monoxide has not been demonstrated.

SOURCES OF CARBON MONOXIDE

A. Emissions Data.

Carbon monoxide is a widely distributed and commonly occurring air pollutant, exceeding all other pollutant emissions combined. Most carbon monoxide emission is from incomplete combustion of fossil fuels. Although large natural sources of carbon monoxide have been identified (Waters, 1972), harmful concentrations have been common only in urban environments near man-made sources. Forest fires may be a major contributor to the total emissions, but there is no data to suggest they increase the recorded ambient concentrations in Montana cities.

In Montana, automotive and industrial sources of carbon monoxide are much higher contributors than natural sources. The Kraft pulp mills and petroleum refinery fluid catalytic cracking units without carbon monoxide controls have relatively high emissions compared to total industrial output in the United States (EPA, 1971). Direct emission data for these plants in Montana is not available.

Emissions prediction data for Billings indicate transportation is the major source there but industrial processes contribute significantly (Ewing, 1971). The data presented by Ewing for 1970, however, does not precisely follow the Stillwater County projections prepared by the EPA (1971 for 1969). Total carbon monoxide emissions for the county were approximately double those projected by the latter authors. This discrepancy resulted primarily from differing estimates of transportation generated pollutants, with the former study predicting higher levels. Estimates for other sources also varied considerably.

Data available for other cities (Ewing, 1971) indicate that forest slash burning and wildfires contribute considerably to the total in western Montana areas. Carbon monoxide data for emissions in Montana are summarized in Table 1.

TABLE I: SOURCE CATEGORY CARBON MONOXIDE EMISSIONS PROJECTIONS FOR MONTANA.

TABLE VALUES ARE IN TONS OF POLLUTANTS/YEAR (1970)

SOURCE:	Great Falls	Helena	Missoula	Billings	Miles City
I. Fuel Combustion	741	939	794	984	683
II. Industrial Process Losses	4,589	-0-	42,052	37,000	-0-
III. Solid Waste Disposal					
A. Open burning	4,914	3,743	4,798	2,420	2,778
B. Slash burning teepee burners	2,686	42,865	216,320	7,286	780
IV. Transportation-Area Sources	131,738	163,669	113,548	133,416	67,579
Miscellaneous Sources					
A. Agricultural burning	2,620	1,345	455	1,350	3,180
B. Forest Wildfires	5,067	57,519	104,438	11,675	3,138
TOTAL FROM ALL SOURCES	152,345	270,080	487,405	194,131	78,138

* References: Ewing et al, 1971DRAFT
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Industrial emissions shown in Table 1 are small compared to the amount put out by automobiles. Because of high automotive emissions, the highest levels of ambient carbon monoxide are usually associated with heavy auto traffic in urban areas.

Emissions from urban home heating fuel combustion also contribute to ambient concentrations. Accumulations of higher concentrations is particularly evident in areas with weather conditions which tend to restrict pollutant movement, as in the Missoula valley.

B. Ambient Data.

High ambient concentrations of carbon monoxide have been associated with concentrated freeway traffic. Recently, Chaney (1978) reported peak concentrations in the Los Angeles area of 45 ppm with average concentrations about 15 ppm. He also reported that fluctuating carbon monoxide concentrations measured inside a moving automobile could be correlated with the passage of other vehicles. Carbon monoxide levels generated by those vehicles varied by a factor of 1 to 100,000. From these data it was concluded that a small number of vehicles produce an inordinately large percentage of the total carbon monoxide. Increased efforts at reducing emissions from these vehicles was recommended.

The high concentrations generally were from older automobiles and heavily loaded vehicles. Recreational vehicles and trucks were also major contributors.

Ambient data for Montana has been collected from three sites in Billings, and one each in Great Falls, Missoula, Butte and Colstrip. Typical data for a selected one month period are depicted in Table III. These data were from busy intersections or traffic routes in Billings, Great Falls and Missoula. The federal nine ppm standard for eight hours was frequently exceeded at the Missoula sampling site (junction of Highway 92 and South Avenue) with violations also recorded at some of the Billings sampling sites on Grand Avenue.

The highest recorded reading was 18 ppm.

Although Montana is not considered to have extensive urban areas, transportation pollution problems are beginning to occur and can be expected to increase with increased population densities.

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TABLE II: TYPICAL AMBIENT AIR CONCENTRATION DATA FOR SELECT MONTANA URBAN AREAS

SOURCE: MONTANA DHES AIR QUALITY BUREAU

COMMUNITY AND COLLECTION SITE	MONTH/YEAR OF COLLECTIONS		MAX. CONCENTRATIONS 1 hr. 8 hr.		VIOLATION 8 HR. STANDARD
Missoula, High.93, Junct.)*	March	1977	17.5	11.1	6
Butte (Alpine West)	March	1977	3.7	2.18	0
Great Falls(908 10th Ave. S.)	December	1977	23.1	14.35	1
Colstrip(McRae Ranch)	May	1975	No readings within detection limits		
Billings (11th and S. 27th St.)*	March	1976	3.9	2.29	0
Billings (Grand Ave.)*	June	1975	9.9	5.47	0

* Violations of the Federal 9 ppm 8 hr. ave. standard are recorded at least once at each of these stations over the monitoring history (Including months other than the data shown).

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HUMAN HEALTH EFFECTS

The effects of carbon monoxide on human health discussed in this review are limited to those which can consistently be measured, in comparison to individuals or populations exposed to lesser concentrations, or none at all. Also considered are effects which tend to single out any significant portion of the population such as those with asthma or heart conditions, who may be particularly susceptible to carbon monoxide.

Because of the hazards of carbon monoxide to human health and the consequent demand for controls on automobiles and other emission sources, many reviews and literature summaries have been prepared. This document includes only the studies believed to be most critical in determining the concentrations which cause adverse effects. Those selected concentrate on the studies related to humans rather than on experimental animals.

Many of the available data refer to "acute," or short term exposures to relatively high concentrations of carbon monoxide. Chronic exposures (long term) are less thoroughly studied. These rely on epidemiological evidence (usually relative health of exposed subjects as compared to subjects exposed to lower ambient concentrations). Because it is very difficult in epidemiological studies to eliminate effects of other variables such as differential occupational exposures, other pollutants and many other aspects, the effects of carbon monoxide are best established in short term controlled studies. These studies address adequately the effects of carbon monoxide on the small group of people being studied but fall short in not addressing the overall question of public health impacts caused both by carbon monoxide alone and in combination with other contaminants. Acute exposure studies, therefore, will be stressed in this review as the basis for determining what ambient concentrations of carbon monoxide have negligible effects on human health.

TABLE IV. AMBIENT CARBON MONOXIDE LEVELS AND ASSOCIATED CARBOXYHEMOGLOBIN IN PERCENT AT EQUILIBRIUM AND AFTER 1 HOUR EXPOSURES*

<u>Percent COHb</u>	<u>Ambient Carbon Monoxide</u>		<u>Heavy Work</u>	
	<u>Light Activity</u>		<u>1 hr.</u>	<u>8 hr.</u>
	<u>1hr.</u>	<u>8hr.</u>		
1.5	21	8	12	8
2	33	11	13	11
2.5	45	14	23	14

* Assumes normal hemoglobin, female at sea level.

Carbon monoxide is highly toxic to humans, and causes many accidental deaths from exhaust leaks into passenger compartments or from running a vehicle engine in a closed garage. It reacts with the red blood cells' hemoglobin to form carboxyhemoglobin. This reaction prevents the transfer of oxygen, which normally is carried by hemoglobin. Because of the resultant decrease in blood oxygen, tissues can be deprived of necessary oxygen, causing tissue death. However, recent investigations indicate toxicity may also be related to carbon monoxide dissolved in the blood plasma, affecting respiration processes in cells by interfering with the biochemical system. This may also explain effects on the nervous system caused by very low levels of carboxyhemoglobin in the blood stream (Goldbaum, Ramirez and Absalon, 1975).

The nervous system is critical in sustaining life and may be the first to show symptoms of carbon monoxide poisoning. All tissues probably are affected by carbon monoxide, and some of these effects may be as important as those to the nervous system. This is particularly the case with the myocardium (heart muscle) when some tissue previously has been destroyed by heart attacks.

Carbon monoxide is most precisely measured in humans and other animals by determining the concentrations of carboxyhemoglobin in the blood. These concentrations indicate the relative dose of carbon monoxide actually in the circulatory system. The relative relationship between ambient concentrations of carbon monoxide and carboxyhemoglobin percent are depicted in Table IV. Use of this table allows a determination of the ambient air concentrations over eight and one hour periods which will result in the carboxyhemoglobin levels reported to produce the various health effects described by authors of the health studies.

Cardiovascular effects have been reported at low doses and currently are the subject of several important investigations. Aronow et al., (1972) have produced the most convincing evidence for measurable adverse effect of very low

concentrations of carbon monoxide. They demonstrated that carboxyhemoglobin levels of three percent speeded up the onset of angina (chest pain associated with impaired heart function in exercising subjects). Anderson, et al., (1973) also provided information regarding the effects of low levels of carbon monoxide on onset and duration of angina. At carboxyhemoglobin concentrations near 2.5 per cent the onset of pain began significantly earlier when the subjects performed exercise. Five and one-half percent carboxyhemoglobin also produced significant increases in pain duration.

Evidence of the damage caused by carbon monoxide to the myocardium was presented by Thomsen and Kjeldsen (1974). Cohen, et al., (1969) provided epidemiological data which indicated correlations between increased mortality from heart attack with increasing levels of carboxyhemoglobin. These data were not definitive in producing conclusions on the effect of carbon monoxide alone, however.

The other important effect of low levels of carbon monoxide involves the decrease in the maximum amount of oxygen that can be taken under stress (maximal aerobic capacity) by individuals who have been exposed to carbon monoxide. The studies of Horvath (1973) indicate definite decreases in maximum aerobic activity. This has not been correlated to reduced athletic ability of young male athletes (cited in Goldsmith and Cohen, 1969).

The very low levels associated with physiological impairment of humans has prompted the following comments by Bartlett (1973): "The impairment of O₂ oxygen delivery by CO is a continuum ranging from severe impairment with high concentrations down to no impairment with zero concentration in the inspired air. There is no evidence for a threshold CO concentration below which there are no effects. The question is not how low a concentration we must achieve to avoid impairment, but rather how much impairment are we willing to tolerate. Clearly, however, people with coronary artery disease are adversely influenced by very low concentrations of CO. Such people represent a sizeable fraction of the population, and we have a responsibility to minimize these risks."

In addition to the studies with humans, many pollution effect investigations have used experimental animals to study acute and chronic exposures, particularly with higher doses of the substances. These studies do not provide any new data important in establishing an ambient air quality standard, so detailed discussions are not included in this review.

ENVIRONMENTAL EFFECTS

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A. Odor and Visibility.

Carbon monoxide is an odorless and colorless gas, so impacts on the aesthetics are minimal. However, it is associated with other combustion generated pollutants that decrease visibility.

B. Agriculture and Other Plants.

Carbon monoxide effects on plants have been observed only at concentrations substantially greater than those known to be harmful to human health. Because of the limited adverse effects on plants, they will not be discussed further.

C. Livestock and Other Animals.

Impacts on livestock and other animals from exposure to ambient carbon monoxide can be expected to parallel human health effects. Although many investigations have addressed the effects of carbon monoxide on experimental animals such as dogs and rats in laboratory tests, field investigations on domestic or wild animals have not been reported.

Lacking other information, it may be assumed that concentrations that do not endanger human health would not produce adverse impacts on livestock or wildlife.

D. Ecosystems.

Carbon monoxide apparently does not accumulate in the atmosphere on a global scale. Current estimates of the half life of carbon monoxide (time required to

reduce carbon monoxide concentrations by half) in the atmosphere range from .1 to 3 years (EPA, 1971). There are apparently "sinks" which cause its continual removal by biological, chemical or physical means.

Because carbon monoxide does not accumulate, and the effects at present ambient concentrations are primarily related to human health problems, no significant effect on ecosystem level phenomena is probable.

E. Materials.

No effects on materials have been recorded, probably because of the relative chemical inertness of carbon monoxide.

Carbon Monoxide Measurement Methods

Measurement - Ref. CFR 40, Part 50, Appendix C

Absorption of infrared radiation by carbon monoxide at characteristic frequencies is the basis for this pollutant measurement. Any carbon monoxide introduced into the sample cell will absorb radiation, which reduces cell temperature and pressure levels by displacing a diaphragm. This displacement is detected electronically and amplified to provide an output signal.

The Montana Air Quality Bureau's experience with the EPA reference method for carbon monoxide has shown it to be excellent. The monitoring devices operated by the state have proven to be very reliable, easy to operate and to calibrate.

CONCLUSIONS

Carbon monoxide is of primary concern to human health. Because most of its effects at low levels have been related to the health problems of a small percentage of the population, the primary question is what percentage or segment of the population should be protected. Individuals with cardiovascular disease appear to be most sensitive to the effects of low levels of carbon monoxide and

these individuals make up a significant percentage of the population. Further, all members of the population apparently are affected by reduced aerobic capacity from carbon monoxide exposure, thus limiting performance in athletic endeavors or other strenuous physical activity.

The effects caused by low carbon monoxide concentration on the central nervous system have not been consistently observed by the various investigators. Although these effects possibly could result in increased automobile accidents, evidence in this regard is not definitive.

There are various sources of carbon monoxide in Montana, but high concentration appear to be related to urban area transportation. Carbon monoxide is a relatively inert compound. This inertness can lead to buildup of carbon monoxide concentrations in stagnant air masses which often are seen in western Montana.

In Great Falls carbon monoxide concentration peaks follow traffic peaks by about two hours. In Missoula this same phenomena is observed, but in Missoula fewer cars generate carbon monoxide concentrations almost double those in Great Falls. On stagnant days in the vicinity of well-traveled streets, carbon monoxide will last longer and influence greater areas than under well-ventilated conditions. Concentrations currently exceed the federal standard for eight hours averages in the vicinity of busy roads and intersections in urban areas. Although other sources such as agricultural burning, forest slash burning, and industrial processes contribute large amounts of carbon monoxide to the atmosphere, no ambient data is available to indicate problem concentrations resulting from these activities.



CARBON MONOXIDE

ALABAMA - 8 hr. 30 ppm (34 mg/m³)

ALASKA - 8 hr. 10 milligrams 1 hr. 40 milligrams

ARIZONA - 1 hr. 40 milligrams, 8 hr. 10 milligrams

CALIFORNIA - 3 hr. 160 ug/m³ 0.24 ppm 12 hr. 10 ppm 11 mg/m³ 1 hr. 40 ppm 46 mg/m³

COLORADO - 50 ppm^a 55 mg/m^{3b'}

CONNECTICUT - 10 milligrams (9 ppm) max. 8 hr., 40 milligrams (35 ppm) max. 1 hr.

DELAWARE - 8 hr. 8 parts pr. million 9.2 milligrams 0.1%

FLORIDA - 10 milg. (9 ppm) max. 8 hr., 40 milg. (35 ppm) 1 hr.

GEORGIA - 40 mill. 1 hr. or 10 milligrams for an 8 hr. 25°C and 760 mm.Hg.

HAWAII - 40 milligrams 1 hr. 10 milligrams for 8 hr. 25°C and 760 mm.Hg.

IDAHO - 10 milligrams (9 ppm) max. 8 hr., 40 mill. (35 ppm) 1 hr.

ILLINOIS - 8 hr. 9 ppm. 1 hr. 35 ppm.

INDIANA - 10 Mill. 10,000 ug/m³ (9 ppm) 8 hr., 40 mill. 40,000 ug/m³ (35 ppm) 1 hr.

IOWA - 17 mill. (15 ppm) 8 hr.

KENTUCKY - 10 mill. (9 ppm) 8 hr., 40 mill. (35 ppm) 1 hr.

LOUISIANA - 10,000 ug/m³ or 9 ppm 1 8 hr. 40,000 ug/m³ or 35 ppm 1 hr.

MAINE - 8 hr. 10 mill. 1 hr. 40 mill.

MARYLAND - 8 hr. mg/m³ 10 ppm, 9 mg/mx0.873=ppm

MASSACHUSETTS - 10 ug/m³ 9 ppm 1 hr. max. 40 mill ug/m³ 35 ppm.

MICHIGAN - 40 ppm (46 mg/m³) 8 hr.

MINNESOTA - 9 ppm 10 milligrams 8 hr.

MISSOURI - 15 ppm 8 hr.

MONTANA - 10,000 ug/m³ 9.0 ppm 8 hr., 40,000 ug/m³ 35.0 ppm 1 hr.

NEVADA - 8 hrs. 10,000 ug/m³ 9.0 ppm; 1 hr. 40,000 ug/m³ 35 ppm; 8 hr. 6,670 ug/m³
6.0 ppm

NEW HAMPSHIRE - 8 hr. 9 ppm, 10 milligrams; 1 hr. 35 ppm, 40 milligrams.

NEW JERSEY - 8 hr. 9 ppm, 40 mill. (35 ppm)

NEW MEXICO - 8 hr. average 8.7 ppm; 1 hr. avg. 13.1 ppm

Carbon monoxide cont'd

NORTH CAROLINA - 10 mill. (9 ppm) 8 hr.; 40 mill. (35 ppm) 1 hr.

NEW YORK - 8 hr. (9) 24 hr.; 1 hr. (35) max. 24 hr. avg.

NORTH DAKOTA - 10 mill. (9 ppm) 8 hr.; 40 mill. (35 ppm) max. 1 hr.

OHIO - 8 hr. 10 mill. 9 parts million

OREGON - 10 mill. 8.7 ppm, 8 hr.; 40 mill. 35 ppm 1 hr.

PENNSYLVANIA - National Standards

SOUTH DAKOTA - 8 hr. 40 mill. 1 hr.

VIRGINIA - 10 mill. (9 ppm) 8 hr.; 40 mill. (35 ppm) 1 hr.

WASHINGTON - 8 hr. 10 mill (9 ppm); 1 hr. 40 mill. 35 ppm

SOUTH CAROLINA - 1 hr. 40,000;; 8 hr. 10,000

TENNESSEE - 10,000 (9.0) 8 hr. ug/m^3 ppm

TEXAS - $43 \text{ mg}/\text{m}^3$ (38 ppm) 8 hr.; $65 \text{ mg}/\text{m}^3$ (56 ppm), 4-hr. or $180 \text{ mg}/\text{m}^3$ (94 ppm) 1 hr.

UTAH - 9 ppm 8 hr. 35 ppm 1 hr.

VERMONT - mg/m^3 ; $10 \text{ mg}/\text{m}^3$ 8.7 ppm 8 hr. 35 ppm 1 hr. $40.3 \text{ mg}/\text{m}^3$

WISCONSIN - 10 mill. (9 ppm) 8 hr.; 40 mill. (35 ppm) 1 hr.

WYOMING - 10 mill. (9 ppm) 8 hr.; 40 mill. 35 ppm 1 hr.

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